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Section of Otology

President—E. G. COLLINS, F.R.C.S.Ed.

[*November 4, 1955*]

The Preservation and Restoration of Function in Otology

PRESIDENT'S ADDRESS

By E. G. COLLINS, M.B., Ch.B., F.R.C.S.Ed.

"TO-DAY, he can discover his errors of yesterday and to-morrow he may obtain light on what he thinks himself sure of to-day."

This is a quotation from the Oath and Prayer of Maimonides, a Jewish physician of the twelfth century.

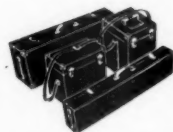Many of us might consider that we are living in one of the most progressive eras of our specialty but let us approach any medical subject with humility for, as Maimonides said, "Knowledge is immense and the spirit of man can extend infinitely to enrich itself daily with new requirements". Many of the accepted facts of to-day may in time become the errors of yesterday. Especially is this true of the function of hearing, about which considerable controversy still exists. Nevertheless, I would like to consider in what manner otology in Britain is fulfilling its social obligation to preserve and restore function.

THE EXTERNAL EAR

A chronic hypertrophic eczematous condition of the outer ear which causes marked stenosis of the meatal canal has frequently proved in my hands to be a most difficult problem. Though temporary improvement may result from various forms of treatment, relapses are common and the otologist is again confronted with a pin-hole meatal canal. I have tried the effect of X-ray therapy with disappointing results. Due to the accumulation of wax, epithelial scales and usually some scanty, thick pus, considerable deafness is present especially if the condition is bilateral. During the last few years I have operated on four such cases with improvement in hearing. The operation was a type of endaural attico-antrotomy with removal of hypertrophic skin, the cavity then being lined by a split-thickness skin graft. Occasionally an unsuspected chronic suppurative otitis media is found and this can be dealt with at the same time. The results are better in the dry, scaly, eczematous type than in the moist type and operation in the latter should only be undertaken in a quiescent phase. Perichondritis did not occur as a complication but operation was always carried out under cover of penicillin. The following are some illustrative cases:

Case I.—Mrs. M. N., aged 51. This patient had a scaly, eczematous condition of the meatal canal for twenty years, without any involvement of the auricle. She had become one of the chronic out-patients at the hospital. Both meatal canals would only admit the finest probe and considerable deafness was present. (Figs. 1 and 2.)
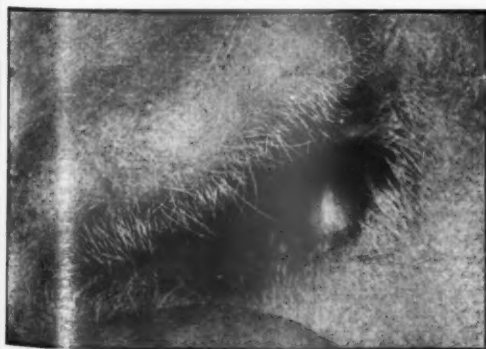

FIG. 1.—Mrs. M. N.
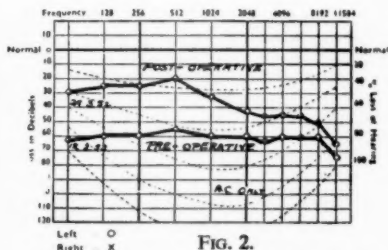
FIG. 2.

FIG. 1.—Mrs. M. N. Post-operative enlarged meatal canal. Lateral view through dissecting microscope (×6) with patient recumbent.

FIG. 2.—Mrs. M. N. Pre- and post-operative audiogram.

Case II.—Mrs. I. R., aged 49. There was a nine years' history of pin-hole meatal canals with involvement of the auricle in the hypertrophic eczematous condition. The post-operative meatal canal is rather more slit-like than in the first case but, nevertheless, considerable improvement in hearing was obtained (Figs. 3 and 4).

FIG. 3.
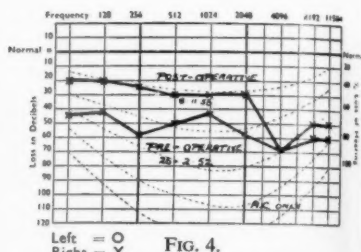
Left = O
Right = X   FIG. 4.

FIG 3.—Mrs. I. R. Post-operative enlarged meatal canal. Similar position to Fig. 1. Note involvement of external ear.

FIG. 4.—Mrs. I. R. Pre- and post-operative audiogram.

Another obstructive lesion which may cause deafness is exostosis of the meatal canals. The subject was extensively reviewed in a paper by Stirk Adams in 1951. Unless the exostoses are large and bilateral, few patients require operation. I have only had to operate on 2 patients of this type, who have both obtained improvement in hearing. It is important that the ivory exostosis should be removed by a burr instead of a gouge as I have seen one patient with a temporary facial paralysis owing to splintering of the bony wall of the facial canal when the gouge was used.

The third obstructive condition of the external ear which may cause deafness is congenital atresia of the meatal canal, though whether this should be classed as an external or middle ear lesion depends on the pathology. In bilateral cases, if the otologist after careful investigation is convinced that he can improve the hearing, operation should be undertaken preferably about the age of 4 or 5, by which time an accurate estimate of the hearing can be made. Whether the unilateral case should be submitted to operation is debatable. The consensus of opinion in this country is against operation if the other ear has normal hearing but Shambaugh (1952) considers that with the improvement in technique made during the last few years, operation even in unilateral cases may be justified. It is a mistake to underestimate the psychological effect that the absence of a meatal canal has in a child and the following case which was operated upon by one of my colleagues illustrates this:

Case III.—R. A. This boy was aged 9. He had bilateral congenital atresia of the meatal canals and the rather callous behaviour of other school-fellows who teased him about his ears had caused him to become isolated, shy and introspective. After operation his mother states his character was completely different. His whole demeanour changed and he now mixed freely with his friends. Considerable improvement in hearing has resulted from operation. A conversational voice is now heard in either ear at a distance of 22 feet; whereas before operation it was about 12–15 feet (Figs. 5 and 6).

The operation I would advocate is a type of endaural attico-antrotomy with skin grafting of the cavity. Magnification should be used and a careful watch kept for any abnormal course of the facial nerve. Often the malleus and incus are found to be fused and, in that event, they may have to be removed. Whether a subsequent fenestration operation is carried out at a later stage depends on the mobility of the stapes which can be gently tested at the first operation.

Where there is microtia present in addition to the congenital atresia the fashioning of the external ear is a tedious and difficult procedure. Any plastic operation to the external ear is better left until the patient is older if it is to be undertaken. After viewing the final result, one sometimes wonders whether the patient might not be better with a prosthesis. The following case illustrates the second stage in the plastic procedure.

Case IV.—Miss V. H., aged 19.   After the meatal canal had been formed, the deformed external ear was divided in its middle and the parts stitched above and below.   A trapdoor type of flap, taken from the skin over the mastoid, was stitched to the edge of the meatal canal.   A pedicle flap was later swung up from the neck to form the remainder of the rim of the helix.   Unfortunately, that is as far as I have been able to proceed with this patient up to the present as she has married and has a young baby, but I do intend to insert some rib cartilage to strengthen the helix, and then at a subsequent stage I intend to provide a posterior skin flap for the ear (Fig. 7).

FIG. 5.

FIG. 7.
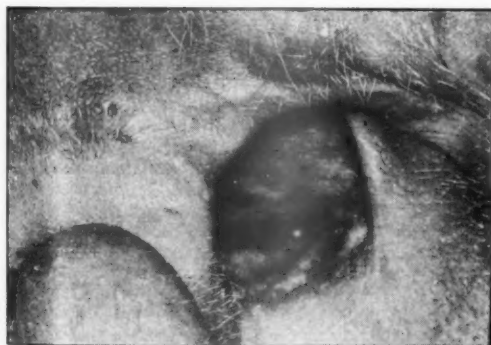
FIG. 6.

FIG. 5.—R. A.   Pre-operative view of external ear.   Small piece of paper marks dimple of external auditory meatus.

FIG. 6.—R. A.   Post-operative meatal canal.   Photograph taken through dissecting microscope (×6).

FIG. 7.—Miss V. H.   Second stage of plastic operation on ear.

THE MIDDLE EAR

The Acute Ear

In 1948 Young and Simson Hall presented a paper on penicillin treatment in acute otitis media with special reference to long-term hearing.   On the whole, the result of this investigation was reassuring but with the use of massive doses of antibiotics there would appear to be an increased tendency to rely on the antibiotics and to discard paracentesis even if the drumhead is bulging.   I am sure this is a wrong treatment.   It is my opinion that if paracentesis is omitted when the tympanic membrane is bulging, the long-term results in hearing will frequently suffer.   This point was not specially investigated either in Simson Hall's series or in Wilson's series of cases reported in 1946, though it was discussed.

Although the majority of organisms in acute otitis media are penicillin-sensitive there is clear evidence that the strains of penicillin-resistant staphylococci are increasing and, if there is delayed resolution of a middle ear inflammation, there is certain to be a greater liability for organization of the exudate with the formation of adhesions.   I have been considerably

impressed with papers by Lumio (1951) and Ojala (1953) on the pathogenesis of chronic adhesive process, and absence of early drainage of an inflamed middle ear is mentioned as one of the causes.

The Chronic Ear

Good hearing, as far as the middle ear is concerned, depends on three factors. Firstly, an intact, mobile tympanic membrane; secondly, a complete functioning ossicular chain and lastly, an unimpaired air space to the round window membrane in continuity with the eustachian tube. Such a statement is a truism familiar to all otologists but it merits emphasis since no benefit will result from any tympanoplasty for chronic suppurative otitis media if the eustachian tube is stenosed, and harm rather than good may result from a fenestration operation for chronic adhesive process if no true fixation of the footplate of the stapes is present. If we intend to adopt a conservative and remedial attitude to our middle ear surgery, we must have a good idea of the effects of middle ear pathology on physiological acoustics and considerable help and guidance can be obtained from the book by Wever and Lawrence (1954) on this subject. Nevertheless, it is not always the case that physiological experiments are in complete accord with clinical experience. For example, Wever and Lawrence found that interruption of the ossicular chain by removal of the incus in the cat, resulted in an average of between 50–60 decibel loss as measured by cochlear potentials whereas clinical experience would rate the loss as between 20–30 decibels in the majority of patients. Another apparent contradiction is that although, clinically, we consider adhesive changes in the neighbourhood of the round window to be of considerable importance in the causation of deafness, cases have been recorded, e.g. Hallpike and Scott (1940), where there has been almost complete closure of the round window by a variety of pathological conditions and yet the patient had apparently normal hearing. Such anomalies are difficult to explain on any resonance theory of hearing and the subject of the round window is especially complex.

Non-suppurative Chronic Otitis Media

I am indebted to Miss Edith Whetnall for the following figures. Of 367 cases of deafness (congenital and acquired) examined at the Queen Mary's Hospital for Children during the last four years, there were 11 cases of chronic adhesive process (i.e. 3%). At another audiology unit where auditory training is given to children, among 800 unselected cases of deafness, 309 were cases of acquired deafness. Of the 309 cases of acquired deafness, 19 were cases of chronic adhesive process (i.e. approximately 6%).

It would consequently appear that the incidence of chronic adhesive process as a causal factor of deafness among children, although not high, is nevertheless of sufficient importance to warrant greater attention being paid to this disease.

Suppurative Otitis Media

Conservative surgery for chronic suppurative otitis media is fashionable and rightly so. Nevertheless, we must retain a balanced attitude to conservatism. If, by substituting some form of attico-antrotomy for a radical mastoidectomy we have not eradicated the pathological condition in the middle ear and have not left the patient with useful hearing, we have failed in the primary object of our operation. On the other hand, I do not believe that conservative surgery is incompatible with a "safe" ear for the patient. If a chronic suppurative otitis media can still be termed dangerous after a conservative operation, then the fault lies either in our selection or in our technique and it is no use blaming conservative surgery. Operative treatment for chronic otitis media which has as its object the preservation of hearing demands more judgment and skill than the majority of fenestration operations. Fortunately we have now at our disposal three useful aids: magnification, the audiometer and the antibiotics.

The more widespread use of magnification in mastoid surgery is the direct result of the fenestration operation and it has certainly enabled us to appreciate more readily the limits of the pathological disease present. The audiometer has enabled us to assess pre-operatively whether conservative surgery is likely to be of value in the preservation of useful hearing and whether the ossicular chain is likely to be functioning. The advent of the antibiotics has removed the necessity for many labyrinthine operations, translabyrinthine drainage and operations on the petrous apex which all involved destruction of hearing. It has also proved of inestimable value in the treatment of intracranial complications. In the James Yearsley Memorial Lecture, Cawthorne (1955a) stated that a radical mastoid operation should be carried out in all patients with intracranial complications resulting from chronic suppurative otitis media and submitted arguments in support of his view. Most of us will agree that a radical mastoid operation is very often necessary, but, personally, I consider that there are cases where a more conservative type of surgery is justified and that each case must be judged on its merits. The keynote to any attempt at conservative surgery is, however, quoted verbatim from this lecture: "If the otologist decides to do anything short of a radical mastoid operation, then he should be prepared to keep the patient under personal supervision". I am in entire agreement with Cawthorne about the necessity for exenteration of a "dead"

labyrinth in a case of meningitis, as, even in these days of antibiotics, I also have seen patients with recurrent attacks of meningitis due to the "dead" labyrinth being left.

In the fascinating papers on tympanoplasty read before this Section earlier this year by Professors Zöllner (1955) and Bocca and Pietrantoni (1955), Professor Bocca described tympanoplasty as a revolution in conservative surgery for chronic otitis media. We must give full credit to Zöllner (1951), Wullstein (1954) and their associates for initiating what is likely to be a most progressive advance in this type of surgery but let us also remember the many otologists throughout the world who have made important contributions. Among former British otologists we might mention the work of Charles Heath, Ernest West and G. J. Jenkins who were pioneers in the concept of preserving function in the operative treatment of chronic suppurative otitis media, whilst among present-day British otologists it would seem right to record the work of Tumarkin (1947), Daggett (1949), McGuckin (1953, 1955), Thorburn (1954) and Dingley (1955) who have given this problem special study. All have played their part in allowing us to reach an intelligent and informed opinion on this subject. Especially would I emphasize the opinion of Thorburn (1954) and Dingley (1955) that if we are to preserve hearing in patients who have a chronic suppurative otitis media with attic or posterior marginal perforations, operation must be carried out early in the majority of patients and we cannot afford the delay of instituting so-called "conservative" local treatment if it involves keeping the patient under observation for months and sometimes years, since we finally reach the stage where conservative surgery is not possible. By this, I do not mean that conservative local treatment has no place whatsoever in the treatment of these types of chronic suppurative otitis media. It is all a matter of clinical judgment and assessing improvement or deterioration of the diseased condition in the middle ear. This can prove extremely difficult.

Dingley (1955) has posed the question as to how it is that the intact pars tensa and malleus exercise a useful function in the preservation of hearing after the ossicular chain has been broken by the removal of the incus. I also find the explanation of this difficult in the light of the physiological experiments of Wever and Lawrence, as they found removal of the tympanic membrane and malleus increased the hearing in the cat after the ossicular chain had been broken by separation of the incudostapedial joint. Although it may not have any sound physiological basis, it seems to me that the good hearing must be related in some way to the maintenance of the air pressure on the round-window membrane. It is only by some such hypothesis as this that one can explain the good hearing results that are being recorded after a tympanoplastic operation in which the ossicular chain is absent. In support of this opinion I would cite the case of an Army officer upon whom I had to perform a radical mastoid operation. A false membrane formed over the middle ear, as was the case in some of Thorburn's most successful radical mastoids, and the officer succeeded in persuading several Medical Boards that his hearing was so good that there was no necessity for his being downgraded.

I do not intend to discuss the various conservative types of operation that may be carried out for chronic ear surgery but I was particularly interested in Zöllner's and Bocca's accounts of the closure of large central perforations by tympanoplasty. For many years I have tried various methods of closure which have been described in the literature with rather indifferent success. The tympanoplastic method does offer a new and hopeful line of approach.

Otosclerosis

On reading Cawthorne's Dalby Memorial Lecture (1955b), I was surprised that he had found the high proportion of 47% of patients with otosclerosis among 2,000 patients who attended a Hearing Aid Clinic and I thought it might be of interest to work out the figures at the Aberdeen clinic.

An analysis of 1,000 unselected cases of deafness in adults who attended the Hearing Aid Clinic at Aberdeen Royal Infirmary was made by my Registrar, Dr. J. L. Doig. I would like to compare these on a percentage basis with the figures quoted by Mr. Cawthorne. All patients had a full clinical examination by a Senior Registrar and all had audiometer tests.

TABLE I.—ANALYSIS OF UNSELECTED CASES ATTENDING A HEARING AID CENTRE

	Cawthorne		Collins	
	Percentage	No. of cases	Percentage	No. of cases
Conductive deafness ..	56·25	1,125	45·1	451
Perceptive deafness ..	35·55	711	33·5	335
Mixed deafness	8·2	164	21·4	214
	100	2,000	100	1,000

There are approximately 11% more cases of conductive deafness among Cawthorne's series and a counterbalancing item of approximately 13% more cases of mixed deafness among Collins' series.

TABLE II.—ANALYSIS OF CASES OF CONDUCTIVE DEAFNESS AND MIXED DEAFNESS

	Cawthorne		Collins	
	Percentage	No. of cases	Percentage	No. of cases
Otosclerosis of all types..	47	943	27·6	276
Other forms of deafness..	53	1,057	72·4	724
	100	2,000	100	1,000

These figures have resulted from an analysis of the cases of conductive deafness and mixed deafness which can be broken down as follows:

TABLE III.—CONDUCTIVE DEAFNESS

	Cawthorne			Collins		
	% Conduction deafness	% Total 2,000	No. of cases	% Conduction deafness	% Total 1,000	No. of cases
Bilateral C.S.O.M. ..	22·3	12·55	251	47·0	21·2	212
Otosclerosis	77·0	43·3	866	52·8	23·8	238
Otitis externa and others	0·7	0·4	8	0·2	0·1	1
	100	56·25	1,125	100	45·1	451

To this figure for the incidence of otosclerosis has to be added the percentage of otosclerosis found among cases of mixed deafness.

TABLE IV.—MIXED DEAFNESS

	Cawthorne			Collins		
	% Mixed deafness	% Total 2,000	No. of cases	% Mixed deafness	% Total 1,000	No. of cases
Otosclerosis and C.S.O.M. ..	22 ⎤	1·8 ⎤	36	12·1 ⎤	2·6 ⎤	26
Otosclerosis and perceptive ..	25 ⎦ 47	2·05 ⎦ 3·85	41	5·7 ⎦ 17·8	1·2 ⎦ 3·8	12
C.S.O.M. and perceptive ..	53	4·35	87	82·2	17·6	176
	100	8·2	164	100	21·4	214

The proportion of otosclerosis combined with chronic suppurative otitis media or perceptive deafness which is related to the total *number of cases examined*, is remarkably similar in the two series (viz. 3·8) BUT the proportion of such cases related to the *total of mixed deafness* differs enormously.

This difference arises from the far larger percentage of C.S.O.M. with perceptive deafness found in Collins' series compared with Cawthorne's series.

These tables indicate that chronic suppurative otitis media (C.S.O.M.) was a causal factor of deafness in a far higher proportion of patients that attended the Hearing Aid Clinic in Aberdeen than was the case at Mr. Cawthorne's Hearing Aid Clinic. Otosclerosis as a causal factor of deafness was considerably less at the Aberdeen Hearing Aid Clinic than at Mr. Cawthorne's clinic. It is agreed that a discrepancy may arise in the assessment of cases of mixed deafness, though all but three of the cases who are classed as chronic suppurative otitis media and perceptive deafness had a clear history of discharging ears. Let us assume that the diagnosis of mixed deafness at the Aberdeen clinic was *not* made on the same basis as at Mr. Cawthorne's clinic, and that the incidence of otosclerosis among the cases of mixed deafness at the Aberdeen clinic was, in fact, as high as that in Mr. Cawthorne's series, namely 47%. Even on this assumption there would only be an increase in the total incidence of otosclerosis at the Aberdeen clinic from 27·6% to 33·9% which is still far removed from Mr. Cawthorne's total incidence of 47%.

The explanation for these discrepancies may be:

(1) That there is a variation in the incidence of the two diseases in different parts of the country.

(2) That there is a degree of selectivity on the part of the family practitioner in advising the patient to attend a specified London hospital where the fenestration operation is very widely practised even though the patient may not be suitable for such an operation and may instead be fitted with a hearing aid.

It is true that Mr. Cawthorne has made every effort to avoid bias but it is difficult to read what is in the mind of a patient with deafness, and every otologist is familiar with the old lady of 60 or 70 who attended his clinic in the early days of the fenestration operation with a demand for an operation to restore her hearing. Probably an accurate estimate of the

incidence of otosclerosis can only be made by obtaining comparable figures from various centres throughout the country.

With justification the otologist might point to the operative treatment of otosclerosis as one of the most significant advances in the restoration of hearing made during this century, for few will deny that the fenestration operation has come to stay. Though there has been a revival in attempts to mobilize the stapes, notably by Rosen (1953) in America, it seems improbable that this operation will displace the fenestration operation, although from the purely physiological aspect it has its appeal. The criticism has been levelled at the fenestration operation that it merely by-passes the disease but Simson Hall (1951) maintains that this criticism is probably unjustified. From his examination of specimens of otosclerotic bone obtained from the living subject, he is of the opinion that otosclerosis can pass from the vascular, active stage, to the healed, quiescent stage. If this is so and otosclerosis really does reach this quiescent stage, then in the majority of patients a successful fenestration operation could be classed as truly curative. Unless we accept this possibility it is difficult to explain why the hearing has beeen maintained in such a large percentage of patients whose fenestration operation dates back to what is now a considerable number of years.

Unfortunately efforts to solve the ætiology of this disease during the last twenty years have yielded no fresh incontrovertible evidence. There are few theories enunciated to-day which were not discussed in whole or in part at the turn of the century. Nevertheless, progress in the pathology of this disease has been made and the researches of Ogilvie and Simson Hall (1953) and Cawthorne (1955b) seem to be moving along parallel lines.

Both believe that otosclerosis is an abnormality of the mesenchyme which is predetermined from birth but it is still a matter of speculation what stimulus activates the otosclerotic focus and causes it to lay down new bone. It is almost certainly some blood-borne factor and may be an endocrine. One always had the hope that eventually some form of prophylactic treatment might be possible to prevent otosclerosis but if the present views are correct this hope is receding. Fowler has suggested that a detailed investigation of the hearing in the children of all otosclerotics might yield useful information and it might be that otosclerosis could be discovered earlier than at present.

One other form of operative treatment which has been remarkably successful in restoring function in the middle ear—though it is not the function of hearing—is the operation for facial paralysis. We have indeed been fortunate in having such a skilled exponent of this operation as our Past President (Miss J. Collier) to give us the benefit of her experience and teaching.

THE INNER EAR

Active treatment by the otologist is less likely to improve function in a perceptive deafness than is the case in middle ear deafness. Reliable histological evidence to support the various theories which were advanced for different types of perceptive deafness has been lacking, though considerable progress has been made during the century. A notable example has been the work of Hallpike and Cairns (1938) on the pathology of Ménière's disease, but, even yet, there are many cases of perceptive deafness in which we have no very accurate knowledge of the pathological background. The electron microscope may cause further advances to be made. Suffice it to state that as a general rule when degeneration and atrophy of the organ of Corti have occurred, the deafness in most cases is irreversible. Before that stage is reached some recovery may occur, either as a result of the inherent recovery powers possessed by the tissues themselves, for example in some forms of concussion deafness, or else by the elimination of some toxin, for example, an apical dental abscess causing a toxic neuritis of the VIII nerve. By and large, however, our efforts have mainly to be directed into two channels. The first is prophylaxis and the second is toward making the maximum use of any hearing present.

Prophylaxis in Perceptive Deafness

The reduction in the incidence of many cases of toxic perceptive deafness has corresponded with the decline in such infectious diseases as diphtheria, typhoid and whooping cough, owing to immunization. In addition, there has been a very definite reduction in the virulence of scarlet fever, measles and mumps which has resulted in fewer middle or inner ear complications.

It would also be reasonable to expect a reduced incidence in cases of "congenital deafness" owing to the correction of Rh incompatibility in the blood, the treatment of syphilis with penicillin and the avoidance of contact with German measles by the pregnant mother. Our knowledge of the toxicity of certain drugs on the VIII nerve has also expanded so that a substitute less lethal to the auditory nerve than quinine has been found in the treatment of malaria, whilst we know that a careful watch on the hearing must be kept during treatment with other drugs such as the salicylates, dihydrostreptomycin and arsenic, to mention a few of the commoner ones in use.

Industry is also not without its hazards in producing a toxic neuritis and lead, phosphorus (match workers), carbondisulphide (rubber workers) and some of the aniline dyes (fur and hair workers) may all cause a perceptive type of deafness. For further information, *see* Taylor (1937).

The effect of tobacco and alcohol in the causation of a perceptive deafness seems rather more indefinite. Robin (1952) quotes the investigation of Carroll and Ireland (1935) who found a well-marked "island" dip in 21 of 36 patients with tobacco-alcohol amblyopia. Only 17% had normal hearing in this group compared with 59% in a control group of a similar age. He concludes that in the pathological findings there seems to be a more toxic effect on the blood vessels of the inner ear than in other toxic types of perceptive deafness. This may be caused by overstimulation of the sympathetic nerves.

Two metabolic disorders that merit consideration are hypothyroidism and avitaminosis: Cretinism will cause a perceptive deafness and one does occasionally come across patients with myxœdema who have an early perceptive deafness which apparently shows some audiometric improvement with thyroid treatment though it is difficult to assess how far this improvement is the result of patients' improved mental reactions and how far it may be due to real improvement in a pathological lesion.

The issue of avitaminosis as a cause of inner ear deafness has been clouded to some extent by the experimental findings of Mellanby (1938) and others in animals. Mellanby produced exostoses in the wall of the internal auditory meatus and degenerative changes in the acoustic nerve in animals by a diet deficient in vitamin A, but that is no criterion that vitamin A therapy will have any effect on a perceptive deafness in man nor was such a claim ever made by Mellanby. Among 303 prisoners of war who showed neurological disorders due to avitaminosis Denny-Brown (1947) found only 13 who were deaf. I myself have never been satisfied that I obtained any worthwhile improvement by the treatment of cases of perceptive deafness by massive doses of vitamin A, though improvement has been noted by Lobel (1949), Ruedi (1954) and others.

Noise in Industry

"Civilization is noise. At least modern civilization is. And the more it progresses the noisier it becomes." (McKenzie, 1916). Acoustic trauma caused by industrial noise is a challenge which British otologists have as yet scarcely accepted, but which must be taken up.

The following have been listed by Machle (1947) as the reasons for postponement of its solution:

(1) Ambiguity in the understanding of what constitutes useful hearing and how it should be measured.

(2) The relatively recent development and standardization of satisfactory instruments for measuring loss of hearing.

(3) The attitude on the part of many people that the situation is better left alone since to investigate the problem of noise would merely emphasize and invite attention to a poorly-defined condition.

(4) A strange reaction on the part of workers who avoid the issue in case it will bring loss of employment or adversely affect their tenure of employment.

(5) The question of compensation for loss of hearing from exposure to industrial noise.

This appears to me to be an accurate assessment and it is clear that what must be achieved is a unity of purpose to overcome the problem on the part of business executives, workers, engineers and the otologist.

Already some of the more enlightened and larger industrial firms are aware of the fatiguing effect of noise. The engineers are also fully prepared to play their part and have indeed made some valuable contributions. In a recent article by one of our former Presidents, Air Vice-Marshal Dickson, on "Noise at Work" (1955) the opinion is expressed that a heavy responsibility rests on otologists and that the present time is propitious to reconcile biophysical, clinical and engineering approaches to the problem of deafness resulting from noise. I desire to give my whole-hearted support to this view but I would welcome the opinion of other members of the Section.

If members consider that positive action should be taken by otologists now, then I would like to move later from the Chair that it be remitted to the Council of the Section of Otology to form a committee of otologists to consider this subject in detail with special reference to the most desirable method of measuring hearing function or loss of it. The committee might well work under the auspices of the Medical Research Council and an approach might be made to them. I feel very strongly that it is for otologists to take the initiative in this matter as they have such abundant evidence of the very serious damage that noise can cause.

So far, I have been speaking mainly about prophylaxis. Let us now consider my other point about making the maximum use of any hearing present. The first problem is:

The Management of Deafness in the Young Child

The difficulties in diagnosing deafness in the young child of pre-school age are known to all but although we pay lip service to early diagnosis, how often is the parent told to bring the child back when he or she is a little older either because we have not the time, the training or the facilities to make an early diagnosis. We have certain centres in this country where this problem has been given special study and of these we may be justly proud but there is no network of such clinics on a Regional basis throughout the British Isles. Admittedly, the management of deafness in the young child is not a problem which concerns the otologist alone but once again I would put forward the plea that the otologists take the initiative. I would suggest that, in every region where this subject has not received recognition, the otologists meet and form a committee who will work in liaison with the Education Authorities, the Public Health Authorities, teachers of the deaf and psychologists, so that eventually the solution of early diagnosis and management of deafness in the young before they reach school age may be found. We can obtain guidance from the larger centres. There can be no more rewarding task for the otologist than to advise anxious parents how their handicapped child can become a useful member of the community.

Ménière's Disease

The discovery of the pathological processes responsible for Ménière's disease made by Hallpike and Cairns in 1938 provided a fresh stimulus for further research on labyrinthine disorders which has proceeded unremittingly throughout the world and has resulted in the clarification of the problem. We can feel a sense of pride in the important contributions which have been made by Hallpike, Cawthorne and their associates. Even yet, however, we do not know the ætiology of Ménière's disease with certainty. The theories which claim most attention are the neurovascular and the allergic theory. Probably the neuro-vascular theory has received more support from British otologists than the allergic theory, which is more widely recognized by some of our American colleagues. I am convinced that before many years have passed this problem of ætiology will be solved. Meantime, are we satisfied with our methods of treatment for Ménière's disease?

The attention of both the otologist and the patient is absorbed mainly with the distressing paroxysmal attacks of vertigo and if the otologist can relieve these by either medical or surgical measures, he is apt to ignore the cochlear symptoms. It is my definite impression that, although I may be fortunate enough to relieve the vertigo by some form of medical treatment, the deafness in the affected ear progresses at a greater rate than can be accounted for by the mere passage of time in the vast majority of patients. Then, if medical measures fail to halt the attacks of vertigo and we have to resort to surgical intervention the operation which has given the best results up to the present is Cawthorne's labyrinthectomy, an operation which destroys the last remnant of hearing in the affected ear. Now, our justification for this destructive operation is that the hearing on the affected side has by this time become distorted and of little value to the patient and this statement is true provided the patient has good hearing in the other ear. Even Day (1952), who attempted to preserve the cochlear function by a discriminative use of diathermy, has agreed that in the majority of patients the attempt is not worth while. Nevertheless no otologist can feel entirely satisfied with such a destructive operation, and we must remember that it has been estimated that in about 10% of cases Ménière's disease may be bilateral.

Dissatisfaction with the treatment of Ménière's disease may, in my case, have been unduly influenced by a sense of helplessness that was engendered by two tragic cases which I have seen lately. The first was a teacher who was the main support of the family. In the course of twelve months she became stone deaf in both ears due to bilateral Ménière's disease.

Case V.—Miss C. S., aged 45. Nine years before she consulted me, this patient had a paroxysmal attack of vertigo with some deafness in the left ear which slowly increased. There were, however, no further attacks of vertigo until December 1952. At this time the hearing in the right ear she considered to be good and this was confirmed on audiometer test (Fig. 8). It was, however,
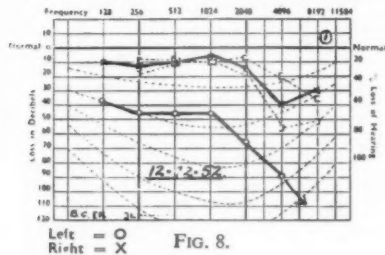

FIG. 8.—Miss C. S. Audiogram at first consultation.

Left = O
Right = X   FIG. 8.

significant that even at this time there was no response to the caloric reactions on either side. The attacks of vertigo continued in spite of treatment and by October 1953 she noticed considerable deafness in the right ear whilst the left ear was practically stone deaf (Fig. 9). After this, deterioration of hearing was very rapid and Fig. 10 is the audiogram made six weeks later.
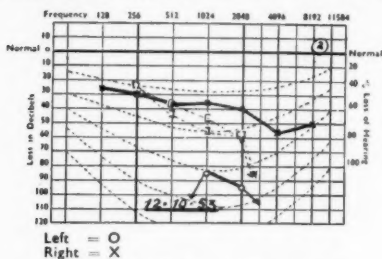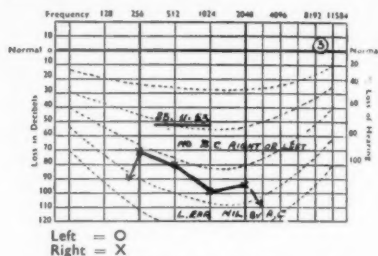

FIG. 9.—Miss C. S. Audiogram ten months later.

FIG. 10.—Miss C. S. Audiogram in a further six weeks.

The Wassermann reaction and all investigations of the central nervous system were negative.

The second patient had a perceptive deafness in one ear from childhood, possibly the result of one of the infectious fevers and now she is suffering from a rapidly progressing deafness in the other ear due to Ménière's disease.

It is cases such as these that make us realize that every avenue which may preserve cochlear function must be explored. Up to the present only Portman's operation on the saccus endolymphaticus and the operation on the cervical sympathetic plexus attempt to influence the pathological process which is present.

My subject of preservation and restoration of function covers such a wide field that, if time had permitted, there are many other problems such as rehabilitation of the deaf, high tone loss in the young child, the problem of increasing presbycusis and psychogenic deafness which might all be included in any critical examination of the progress we have made in preserving function.

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Section of Laryngology

President—W. A. MILL, M.S., F.R.C.S.

[*November* 4, 1955]

Cancer of the Larynx: Laryngectomy after Radiotherapy

PRESIDENT'S ADDRESS

By W. A. MILL, M.S., F.R.C.S.

I THOUGHT it would be useful to go into the results of cases of cancer of the larynx treated by laryngectomy, particularly as most of the cases upon which I have operated have previously been treated by radiotherapy. The number of cases is not large and one must not therefore draw conclusions too readily, but certain definite indications seem to emerge.

From 1933 until the present time well over 500 cases of cancer of the larynx have been seen at the Royal Marsden Hospital alone. Many of these cases have been referred for treatment from other hospitals and after their treatment has been completed they have returned to those hospitals for purposes of follow-up and if necessary for operation. From early 1946 until September 1955, 47 cases of laryngectomy have been under my care at the Royal Marsden Hospital and at St. Thomas's Hospital. One or two of the earlier operations were done by or with the late Mr. Lionel Colledge and several were done by Mr. Peter Huggill. The radiotherapy was given in most cases by Dr. M. Lederman by teleradium at the Royal Marsden Hospital or by Dr. J. A. C. Fleming by deep X-ray therapy at St. Thomas's Hospital and to them I am grateful for their help and co-operation. But other cases have had radiotherapy at other London hospitals or elsewhere in England, and in Canada and India. I must thank many colleagues for referring to me cases requiring operation.

TOTAL LARYNGECTOMY FOR CANCER OF LARYNX (TABLE I)

All cases have been traced and those that are alive are almost all seen regularly at follow-up clinics. 3 of the 47 cases were women. The youngest patient was 35 years old and the oldest 75.

TABLE I.—TOTAL LARYNGECTOMY FOR CANCER OF THE LARYNX

	Number of operations	Survivors	Died of disease	Died within four weeks of op.
Total	47	29	15	3
Over five years after operation	15	9	5	1
Over three years after operation	12	6	5	1
Operation under three years ago	20	14	5	1
		2 with disease		

The results of operations done over five years ago are shown. Of these, of the 9 patients who survived without disease for over five years after operation, 2 have since died. One died of severe bronchitis with no recurrence of carcinoma. He had been badly gassed in World War I. I have seen several such patients who have subsequently developed carcinoma of the larynx. The second died, over five years after operation, of cerebral thrombosis following carbon monoxide poisoning: an open verdict was returned. No growth was found at post-mortem. 2 of the longest survivors are alive and well over nine years after operation.

Patients do not always accept the advice given to them—and sometimes, of course, they prove to be right. However, there was in one or two cases delay in initiating treatment by radiotherapy and later, when laryngectomy appeared necessary, there was further loss of time and this, it is certain, is undesirable.

Table II shows the total of cases done over five years and over three years ago.

TABLE II.—TOTAL LARYNGECTOMY FOR CANCER OF THE LARYNX
Total of cases over 3 and 5 years after operation

Number of operations	Survivors	Died of disease	Died within 4 weeks of op.
27	15	10	2

In Table III appear the results related to treatment received before laryngectomy whether

TABLE III.—PREVIOUS TREATMENT AND RESULTS OF LARYNGECTOMY

	No. of operations	Survivors	H.V.T.		Teleradium		Other operation		No previous operations	
			Alive	Dead	Alive	Dead	Alive	Dead	Alive	Dead
> 5 years	15	9	0	2	8	3 1 P.O. 1 T.	1 F.H.	1 L.F. + ?F.H. + T.	0	
> 3 years	12	6	1	1 T.	4 1 T.	3 1 P.O. 1 B.D. 1 T.	1 L.F.	2 1 L.F. 1 T.	0	
< 3 years	20	14	3	0	8 2 T. 1 disease +	6 1 P.O. 2 B.D. 1 T.	1 L.F.	0	2 1 B.D. Disease +	0

P.O.—Post-operative.   F.H.—Finzi-Harmer operation.   L.F.—Laryngo-fissure operation.

T.—Tracheostomy.   B.D.—Block dissection of cervical lymph glands.   H.V.T.—High voltage therapy.

by radiotherapy or by some previous operation. The numbers are, of course, small but the results in cases previously treated by teleradium are encouraging.

When first seen 6 of the 32 patients treated by teleradium had growths suitable for removal by laryngo-fissure. Of these 5 survive and 1 is dead after subsequent laryngectomy. None of the other 26 patients was suitable for the laryngo-fissure operation.

The average lapse of time from the start of teleradium treatment till laryngectomy was fifteen months. Teleradium treatment would occupy about six weeks in most cases. In cases that died of disease the average interval between the start of teleradium treatment and laryngectomy was nine and a half months and in those that survived seventeen months. The shortest time was three months and the longest was six years.

One case previously treated by H.V.T. recurred after four and a half years. One case recurred after laryngo-fissure four years beforehand.

In one or two cases there was delay in initiating preliminary radiotherapy; one patient in whom a positive biopsy was obtained went to Ceylon for a year before returning for treatment by H.V.T. in October and November 1950. Tracheostomy was done in Ceylon in April 1951, and he did not return for laryngectomy till April 1952. He died of recurrent disease just over a year later.

Post-operative deaths.—In the 47 cases there were 3 post-operative deaths within four weeks of operation. One case, a woman, died of a massive pulmonary embolism twelve days after operation. The emboli came from thrombosed veins in the right calf. 2 patients died of respiratory difficulty. In one case, a Maltese who could not speak English, nursing

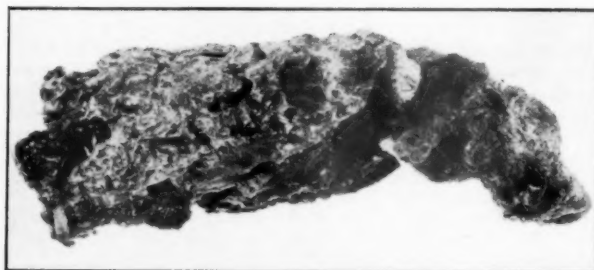FIG. 1.—Crust removed from trachea after laryngectomy (9½ cm. long).

was a great problem. His neck was very short and fat and after the operation became infected so that the stitches of the tracheostomy cut out and the trachea dropped down into the mediastinum. Repeated bronchoscopies to remove crusts were carried out. Some of the crusts removed were of considerable size (Fig. 1). He died twelve days after operation.

There was intense bronchitis and tracheitis with mucopurulent material in the trachea and bronchi. The other post-operative death occurred suddenly at night six days after operation. At post-mortem there was severe bronchitis and pulmonary collapse. A plug of mucus and dried blood was found in the trachea. I feel his life might have been saved by immediate bronchoscopy.

Diagnosis.—The diagnosis of carcinoma of the larynx may sometimes be most difficult. The diagnosis of the recurrence of disease or its persistence after radiotherapy may be awkward because of some degree of laryngeal œdema and perichondritis. Biopsy may be misleading. It would seem that clinical judgment must be trusted rather than a negative biopsy. In one case a patient was seen early in 1947 but he would not attend hospital the following week although strongly advised to do so, and it was not until August 1948 that he did so again. In spite of a negative biopsy the case was considered to be one of carcinoma. Exploratory laryngo-fissure was considered. Again he refused to have any treatment until a tracheostomy became necessary in November 1948. It was only after this that a biopsy was obtained which showed anaplastic carcinoma. Teleradium treatment was unsuccessful and a laryngectomy was done in April 1949. After operation a small fistula developed in the mid-line of the neck. An attempt was made to close it but it became obvious that the fistula was a malignant one. Later secondary deposits in the lung were shown by X-ray examination.

In another case seen with Mr. D. F. A. Neilson no positive biopsy could be obtained until an exploratory operation was performed. Clinically the diagnosis of carcinoma had been made.

In a case which Mr. Lionel Taylor kindly allowed me to see on several occasions, active tuberculous laryngitis and carcinoma of the larynx were present at the same time.

One has seen several cases of tuberculous laryngitis referred for treatment by radiotherapy under the impression that the disease was malignant. Tuberculous laryngitis is much less common than it used to be and its presence is not so readily suspected nowadays.

SITES OF PRIMARY GROWTHS (TABLE IV)

The classification adopted is that described by Dr. M. Lederman (1952, 1954).

TABLE IV.—SITES OF PRIMARY GROWTHS

	Alive	Dead
Supra-glottic	2 (1 disease+)	5
Glottic..	21	4
Sub-glottic	4	5
Epi-laryngeal	2 (1 disease+)	0
Chondro-sarcoma of cricoid ..	0	1
Total	29	15

It will be seen that glottic growths are by far the most common and that the results in these cases are by far the best. In supra-glottic and sub-glottic growths there is much more risk of recurrence.

TABLE V.—HISTOLOGY OF 44 CASES WHO SURVIVED POST-OPERATIVE PERIOD

	Alive	Dead
Anaplastic carcinoma	2	3
Active squamous-cell carcinoma	2	2
Squamous-cell carcinoma	25	9
Chondro-sarcoma	0	1

Histology.—Table V shows the histology in the 44 cases that survived the post-operative period. In most cases the slides reported on are those taken just before or after laryngectomy. In a few cases the reports are those obtained before radiotherapy was given. As is to be expected the results of operation in squamous-cell carcinomata are better than those in anaplastic carcinoma or active squamous-cell growths. Occasionally the report of variably differentiated carcinoma was given. One part of the section may show marked differentiation and keratinization and another part relatively little differentiation. In at least 2 cases no positive biopsy could be obtained by direct laryngoscopy before laryngectomy but both proved to be squamous-cell growths when examined after removal of the larynx. I have felt that on occasions there has been too long a wait for a positive biopsy and one should trust to clinical judgment more readily at times. The piece removed for microscopical examination is often very small and the growth may be missed. On one occasion when

E

there was strong clinical suspicion of malignancy no carcinoma was shown in 6 sections. The whole block was then cut through and carcinoma was demonstrated in 10 out of 30 sections.

In one case of proved squamous-cell carcinoma there appeared clinically to be recurrence on the vocal cord and in the sub-glottic region nearly four years after teleradium treatment. Biopsies from both sites showed "in situ carcinoma". I was tempted to do a laryngo-fissure operation but I did not do so because of the sub-glottic swelling. Examination of the larynx after removal showed only "in situ carcinoma". Of course "carcinoma in situ" (or intra-epithelial carcinoma) may appear in a section when the adjacent area shows infiltrating carcinoma. I think one must be guided by clinical judgment as to what is best for the patient.

In a case previously treated in 1948 for carcinoma of the right pyriform fossa suspicious changes appeared in the right aryepiglottic fold in September 1954. Biopsy revealed "carcinoma in situ". The patient was watched carefully; a change was noticed and two recent biopsies in 1955 showed (1) anaplastic carcinoma and (2) squamous-cell carcinoma and "carcinoma in situ". Laryngo-pharyngectomy was refused. The patient is still under observation and at work as a seaman.

Keratosis or leukoplakia of the larynx.—In only one of the cases was a definite diagnosis of leukoplakia made years before carcinoma developed. A man was seen for several months in 1939 at St. Thomas's Hospital. He had keratosis of the right vocal cord. He then joined the Army and was lost sight of for some years. In 1945 he attended the Westminster Hospital and Professor Ormerod had him under observation for several years. In 1950 the appearance suggested to Professor Ormerod a malignant change. Laryngectomy was refused and so treatment by 10 grams teleradium was given at the Westminster Hospital. The result was not satisfactory but he refused operation. In 1953 he attended St. Thomas's Hospital again and submitted to laryngectomy. He remains well and is at work.

On another occasion one has followed a case of keratosis which has become malignant after a number of years but the history of the above case is the longest that I have met with personally.

It is remarkable how on going over the cases of laryngectomy there is the complaint of hoarseness or huskiness for many years in quite a number. Unfortunately no clinical details are available of the exact cause, but the symptom is suggestive of long-standing keratosis.

In one case of well-differentiated squamous-cell carcinoma which had been treated by the Finzi-Harmer method in September 1945 there was in July 1946 ulceration of the left vocal cord which was completely immobile. There appeared to be no doubt clinically of recurrence of disease and no biopsy was taken. After laryngectomy only ulceration and necrosis were discovered microscopically. Further cutting of the block in September 1955 has failed to reveal any malignant disease. It is possible, of course, that any growth present may not have been taken for microscopical examination.

Another such case is of a patient with proved squamous-cell carcinoma of the whole length of the left vocal cord and anterior end of the right vocal cord treated by teleradium in 1945. Some months later a large mass was seen on the left cord and recurrence was diagnosed. No biopsy was done as the diagnosis of carcinoma did not seem to be in doubt. The microscopical report on the excised larynx failed to show evidence of malignant disease but showed radiation effects with fibrosis and chronic inflammatory-cell infiltration and areas of superficial ulceration.

Cases like these might raise interesting medico-legal points. However, one should as a rule trust to clinical judgment. So often laryngectomy has been delayed for too long because negative biopsies have been obtained or because the appearances in the larynx have been thought to be due to a degree of radiation effect or perichondritis. It is at times very difficult to arrive at the true diagnosis.

Chondrosarcoma of larynx.—The case of chondrosarcoma is one of great interest. Sirota and Hurwitz (1952) in recording a case said it was only the fifth to be reported in the literature.

My case was referred by Mr. Arthur Miller in April 1949. She was a Frenchwoman, a Corsican who taught French in a very well-known girls' school. I am very grateful to Mr. V. E. Negus for his help in establishing the diagnosis. She came with a tracheostomy and with a speaking valve she could continue to teach (Fig. 2). On two occasions (once by Mr. Negus) the tumour was removed as far as possible by the thyro-fissure route. Teleradium treatment was given because for a long time she refused laryngectomy. Eventually she agreed to this and it was done (Fig. 3). She died, however, about nine months later, in August 1952. There was no obvious recurrence in the neck but there were generalized metastases in the lungs, adrenals, pancreas, liver and bones, especially the pelvis.

FIG.
X-ray sh
track

Fig. 2.—Chondrosarcoma of larynx. X-ray showing tumour and tracheostomy track.

Fig. 3.—Chondrosarcoma of larynx excised by operation.

SITES OF RECURRENCE AFTER LARYNGECTOMY (TABLE VI)

Again the number of cases is small and too much must not be deduced from the figures. It would seem, however, that glandular metastasis is less common after radiotherapy than might be expected. In only three cases were palpable glands present at the time of laryng-

TABLE VI.—SITES OF RECURRENCES AFTER LARYNGECTOMY

Site					Alive	Dead
Cervical glands	..	..	..	..	1	4
Tissues of neck	..	..	..	..	1	4
Pharynx	..	..	..	..	0	1
Tracheostome	..	..	..	..	0	4
Generalized	..	..	..	..	0	1
(Necrosis)	..	..	..	..	0	(1)
			Total		2	15

ectomy. In two of these recurrences occurred after block dissection. In the other case a small gland was noticed in the right side of the neck with a growth in the left side of the larynx. It was decided to remove the gland for microscopical examination after laryngectomy. The gland, however, very rapidly became enlarged and fixed. The growth was an anaplastic carcinoma.

In another case which Mr. Lionel Taylor kindly allowed me to see a metastatic gland developed on the opposite side of the neck to the carcinomatous cord. In two other cases where glandular metastases appeared some months after laryngectomy block dissection did not bring about a cure.

It has always been the practice to examine the carotid sheath region for glandular metastases during the laryngectomy operation.

Sometimes glandular metastases yield to radiotherapy. In a case referred by Mr. Lionel Colledge in 1937 there was a large fixed gland 3·5×7 cm. in the left side of the neck after laryngectomy about a year previously. This was treated with the 1 gram radium bomb and the patient remains well to this day. For fifteen years after the operation he earned his living as a foreman in a gas company using an artificial larynx.

Another patient had had his larynx removed by Mr. A. G. Tressider over five years ago. A few months later he developed enlarged glands in the left jugulo-digastric region; one of them 4×3 cm. was hard and fixed. He was treated by teleradium (10 grams) and he remains well and at work to-day.

In neither of these cases had there been treatment by radiotherapy before laryngectomy.

In looking at these results of recurrence one may think that perhaps there should have been a greater readiness to remove the hyoid bone and the whole pre-glottic space. It is, of course, sometimes difficult to say whether the recurrence is glandular or in the *neck tissues*. The figures are given with this in mind. I do not believe in so-called prophylactic block dissection of cervical glands; in the old this operation is very disabling.

In my experience the post-operative treatment of recurrences by teleradium and H.V.T. has been very disappointing. This applies to small recurrences in the mid-line of the neck, recurrences round the stoma and to those in the neck tissues and glands. The treatment of recurrences with interstitial radium needles or "gold grains" has also been unsatisfactory. The 15 cases that died of disease did so, on the average, just under a year after operation.

OPERATIONS BEFORE LARYNGECTOMY (TABLE VII)

The late Mr. Lionel Colledge always emphasized the likelihood of recurrence in cases

TABLE VII.—OPERATIONS BEFORE LARYNGECTOMY

Operation	Alive	Dead	Site of recurrence
Tracheostomy 8	3	5	1 widespread *chr. sarcoma* 3 tracheostome *2 anaplastic* *1 variably differentiated carcinoma* 1 front of neck *anaplastic*
Laryngo-fissure 3 Finzi-Harmer 2	3	2	1 alongside tracheostome *? gland* 1 mid-line of neck

where a tracheostomy (Fig. 4) had been done beforehand, especially if some time beforehand.
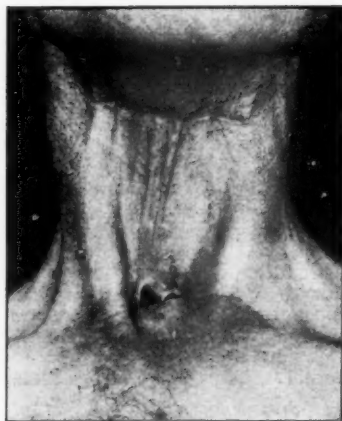
FIG. 4.—Recurrence of carcinoma round tracheal stoma eleven months after laryngectomy. Some months before laryngectomy a tracheostomy had been done.

FIG. 5.—Recurrence, tissues of front of neck, some months after laryngectomy. Patient had a laryngo-fissure operation ten months previously.

It would seem that the figures given here confirm this and possibly the same applies in cases of laryngectomy after the Finzi-Harmer and laryngo-fissure operations (Fig. 5); in these, of course, it is likely that a tracheostomy had been carried out.

Effects of radiotherapy on the tissues.—Professor D. W. Smithers has kindly given me some information about the effects on tissues of teleradium and X-ray therapy.

There are considerable differences between an X-ray beam of 150–200 kV and a teleradium beam as used in clinical practice, although the distribution of the two, as measured in a water phantom, may be very similar.

With the teleradium beam the maximum dose is not on the skin surface but is displaced a few millimetres in depth and so the skin reactions for the same maximum dose of radiation are less with radium than with soft X-rays. A more important difference, however, is due to the varying composition of the patient's tissues; far more energy is absorbed in denser tissues of higher atomic number from the X-ray beam than from the radium beam.

With the radium beam, therefore, there is not only less surface skin absorption but also less absorption in bone and cartilage and so, with more energy left in the beam to be absorbed in the tumour, a higher tumour dose results, for less normal tissue damage.

With supervoltage X-ray therapy, above 1,000,000 volts (where the radiation is of more or less the same quality as radium radiation) these differences disappear.

HEALING AFTER LARYNGECTOMY FOLLOWING RADIOTHERAPY (TABLE VIII)

Of the 44 cases who survived the post-operative period 29 had previously had teleradium treatment and in 22 of these healing was by primary intention. In one case a pharyngostome

TABLE VIII.—HEALING IN 44 CASES OF LARYNGECTOMY

		Primary intention	Temporary leaks	Pharyngostomes
Previous teleradium	.. ..	29	22	6	1
Previous H.V.T...	.. ..	7	1	4	2
No previous teleradium or H.V.T.	8	5	3	0
Totals		44	28	13	3

developed (Fig. 6) and Mr. Alan Hunt closed it by a plastic operation. In 6 other cases

FIG. 6.—Pharyngostome after laryngectomy following teleradium treatment. Appearance four weeks after operation.

a small pharyngeal leak developed usually about the fifteenth day after operation. The leaks closed spontaneously in a week or two, but in one case persisted for five weeks.

Of the 7 cases operated on after deep X-ray therapy 6 developed leaks within the first week or ten days. The leaks closed spontaneously after about four weeks in 4 cases but in the

2 others large pharyngostomes developed.   Mr. R. J. V. Battle, by a series of operations, closed the opening in one case (Figs. 7 and 8).

FIG. 7.—Pharyngostome following laryngectomy after deep X-ray therapy. Appearance two months after laryngectomy.

FIG. 8.—Appearance one year later after several plastic operations by Mr. Richard Battle.

One case operated on after H.V.T. has caused me ever since to approach such cases with caution. First seen in June 1946 and considered unsuitable for laryngectomy he was treated by H.V.T. Owing to a breakdown of the apparatus treatment extended over four months and the tumour received just over 8,000 r. All was well till July 1949—three years later—when recurrence of disease, squamous-cell carcinoma, necessitated laryngectomy. Ten days after operation bilateral pharyngeal fistulæ developed. These increased in size and large pharyngostomes developed. There were repeated hæmorrhages. On one occasion Mr. G. H. Bateman ligatured some large veins. About four months after operation the right common carotid had to be tied because of necrosis of its wall. The following day a left hemiplegia developed and within a month the patient died. At post-mortem there was no macroscopic evidence of growth.

In 8 cases where there had either been no previous treatment or operation by laryngo-fissure or Finzi-Harmer techniques 5 cases healed without a leak and 3 developed small leaks, which closed without difficulty. None developed a pharyngostome. Whilst a lateral leak is not very serious, a leak in the tracheostome may be a great nuisance to the patient.

Disadvantages and Advantages of Operation after Radiotherapy

There is occasional difficulty in healing but really very few serious troubles in operations after radiotherapy, but the operation is more difficult, the skin has become fibrous and so have the fascial layers. On a few occasions the infra-hyoid muscles have been found to be quite white from fibrosis. In my experience a pharyngeal fistula is no more likely to appear after teleradium than in a case operated on without previous radiotherapy.

The advantages in giving radiotherapy are that some will do well with radiotherapy alone and no laryngectomy will be required even in advanced cases. Dr. M. Lederman (1952, 1954) has given the results of teleradium treatment, showing that this is so. No one can say beforehand whether any case, however early, will be cured by radiotherapy or, for that matter, by operation. With early cases small fields are used in teleradium, but in advanced cases with the use of larger fields some of the gland areas will receive radiation incidentally. Then again malignant cells in the lymphatics or in the lymphatic glands may be destroyed by the radiotherapy. In supra-glottic cases treated by radiotherapy the gland areas are treated. At any rate glandular metastasis in the cases reported here does not seem to be of frequent occurrence. There have been several cases with stridor where tracheostomy has been avoided by the giving of radiotherapy first and I am sure this is of great value.

Cases where Laryngectomy was Refused or thought Inadvisable

The following cases are mentioned just as examples of what may happen when the operation of laryngectomy is not done, although it would seem to be indicated.

Just over five years ago a patient with severe tabes and marked lingual leukoplakia developed a squamous-cell carcinoma of the right vocal cord and mouth of the ventricle with complete fixation of the cord. He was treated with H.V.T. and remains well. His work in a London theatre as well as the tabes made laryngectomy undesirable in his case.

Three and a half years ago laryngectomy was advised in a patient with an ulcerated fixed right vocal cord (squamous-cell carcinoma). Operation was refused. Treatment by H.V.T. was given. The cord remains fixed but there has been no recurrence of disease.

Another patient, a woman, was aged 67 when first seen in January 1945. She refused laryngectomy and lived after teleradium treatment till September 1951. She died of the disease at the age of 73 years.

On two occasions the operation has been abandoned because disease had invaded the infra-hyoid muscles. One died a few months later. Another after treatment by H.V.T. is well and at work with a tracheostomy nearly a year later. The growth was a moderately differentiated squamous-cell carcinoma. I think there is probably disease present now. These have not been included in the 47 cases.

OPERATION

It is not intended to go into operative technique in detail but a few points may be mentioned.

Anæsthesia: General anæsthesia is used. Twenty-four years ago I operated on my first laryngectomy under local anæsthesia. The operation went well and healing was uneventful but I have never repeated the experience.

Under general anæsthesia a cuffed intra-tracheal tube is passed through the mouth. When the pharynx is opened the tube is brought out through the pharyngeal opening. When the larynx has been separated it is threaded off the tube (Fig. 9), the intra-tracheal part of the
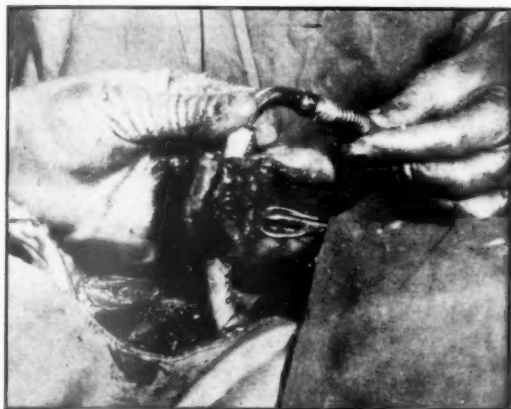
FIG. 9.—Larynx excised and about to be slipped off intra-tracheal tube.

tube and the inflated cuff not being disturbed from the start of the operation until it is ended.

It is sometimes said that this technique encourages recurrence in the trachea or round the tracheostome. I do not believe this. It is most important to keep any blood from reaching the lower part of the trachea and bronchi and I know of no other certain way.

Low blood pressure technique has been used on a few occasions, but I have given it up. On one occasion with hexamethonium bromide the patient was thought to have died; however, he revived and the operation was completed. The patient is well nearly four years later. On another occasion a large empty anterior jugular vein was divided before the vein was recognized, but fortunately the cut ends were seen and ligatured. It might not

always be the case.   Bleeding later or possibly air-embolism might result in such a case. Possibly Arfonad is a safer drug to use than hexamethonium bromide.

As a rule the *Sorensen Flap* is used but sometimes a mid-line incision with upper and lower horizontal incisions is employed, especially if there is the possibility of having to make a pharyngostome at operation or when there is a tracheostomy already present.

There is no doubt that the antibiotics and especially penicillin have brought about improved results.   Intramuscular penicillin is administered for at least five days after operation.   The wound is powdered with penicillin and sulphathiazole powder.   The pharyngeal mucosa is sewn up with two layers of stitches and the inferior constrictor muscles are sutured to make a third layer.   Drains are inserted in each side of the neck and removed on the fourth day. In this way any pharyngeal leak tends to have an oblique course.   At the end of the operation the trachea is inspected and any blood in it sucked out.   The tracheostomy tube used is Moure's modification of Lombard's tube.   The conical part of the tube is well covered with gauze to cork the tracheostomy opening (Fig. 10).   Great care is taken with hæmostasis, especially near the tracheostome.
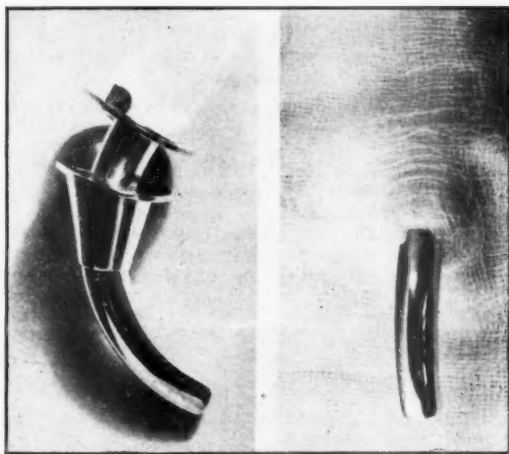

FIG. 10.—Moure's modification of Lombard's tube with gauze arranged to cork tracheostomy opening.

The skin sutures approximating the skin and tracheal mucosa always encircle a tracheal ring and this usually ensures a good stoma.

The trachea should be bared of the surrounding soft tissues as little as possible so as to preserve a good blood supply.

I have, by the way, seen necrosis of the tracheal rings in one case (not included in the 47 cases) of laryngo-pharyngectomy following teleradium treatment of a post-cricoid carcinoma.

Dressings and plenty of wool are bandaged firmly to ensure the obliteration of dead spaces and splinting of the neck.   Sedatives are hardly used and no morphia is given.

The polythene feeding tube is passed through the nose down to about the mid-point of the œsophagus.   It is kept in position by a stitch through the columella of the nose.   If the tube is not stitched it may be coughed out, "come out", or be pulled out by the patient. Even so the stitch has been known to cut out.   If the tube comes out for any reason before the tenth day a pharyngeal fistula usually results.   I have not dared to do without a feeding tube, as is sometimes recommended.

Breathing exercises are given by a physiotherapist before and after operation and the patient is encouraged to get out of bed the day after operation.

Speech.—Of the 29 patients who survived 23 learnt to speak well with the pharyngeal or œsophageal voice.   One is a lawyer still in practice.   Another is a busy surgeon and yet another has acted as a foreman of a gang of steel erectors.   One who can say only an occasional word finds the electric larynx of use, especially in dictating letters—he is a business man.   Most of the others are still having lessons and except for two are making progress.

Disabilities after laryngectomy.—There are occasionally more than the obvious disabilities. Patients hate to catch a cold, which may lead to tracheitis with mucous expectoration and crusting and bleeding.   Alevaire sprayed into the trachea seems to relieve this.

I asked a surgical colleague whose larynx I removed four and a half years ago to give me his experiences and this is what he says:

(1) *Cough.*   Every morning on waking and sometimes during the night one is forced to clear the trachea of secretion and after putting on a shirt and collar there is always some irritation for the first few minutes which forces one to cough.   This cough may recur frequently during the day, especially after talking.

(2) *Involuntary sneeze.*   In some ways the most irritating feature is the *sudden involuntary sneeze* which takes place from time to time during the day and may occur before one can cover the opening with a gauze swab.   I have tried wearing a piece of gauze under the shirt during the day but found this is inclined to get dirty and one cannot clean oneself without replacing it.

(3) *Flatulence.*   Another disability is the *excessive* abdominal flatulence which causes one to wake up practically every night, once or twice, and is sometimes accompanied by colicky pain until the flatus is dispersed.

(4) *Tracheitis.*   When there is an epidemic of colds and an infection is picked up it causes tracheitis.   This sometimes persists for several weeks and the mixture of inspissated mucus and blood which dries up in the trachea is very difficult to dislodge, even with forceps.

One or two other patients have complained bitterly of wind, especially one who has been much bothered by a fistula-in-ano.   Another troubled with hæmorrhoids did not learn to speak well till the hæmorrhoids had been dealt with by operation.

Because of sepsis during healing the stoma may tend to contract.   My colleague Mr. G. H. Bateman had such a case.   Tubes worn to keep the stoma open cause coughing and ulceration and crusting in the trachea with diminution of its lumen.   Mr. Bateman designed a tube (Fig. 11) with a narrow flange to rest well on the stoma between the two sternal heads of the sternomastoid muscles (Fig. 12) and of such a shape and length as to overcome the difficulty.

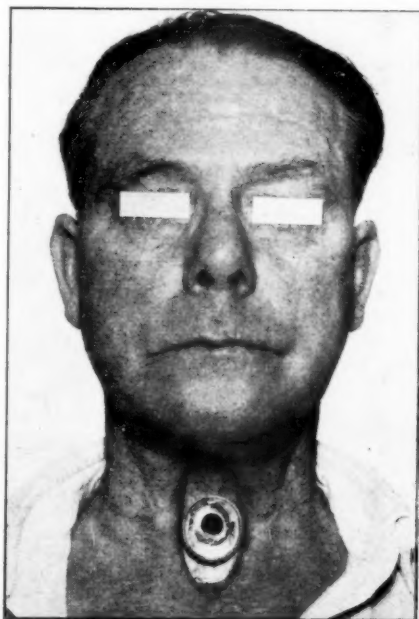Fig. 11.—Tube designed by Mr. G. H. Bateman for wear after laryngectomy.

Fig. 12.—Tube in position.

I would like to mention here a case referred in 1952 by Mr. E. H. Rainer. He had a keratinizing squamous-cell carcinoma of the whole length of the left vocal cord. Treatment by teleradium (10 grams) was given (7,620 r in forty-eight days). The cord returned to normal and has remained so. In September 1954 a small swelling was noticed on the posterior third of the right vocal cord. Biopsy proved it to be a squamous-cell carcinoma. The right cord was removed by laryngo-fissure by Mr. Rainer and healing was quick and satisfactory. When seen recently the patient was very well, with no recurrence.

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MEETING HELD ON DECEMBER 2, 1955

A DISCUSSION was held on **Pharyngeal Paralysis**. The opening papers, read by Dr. FRANK ELLIOTT, Dr. JOHN HARMAN and Mr. CHARLES KEOGH, will be published in the *Journal of Laryngology and Otology*.

The following took part in the discussion: Mr. N. ASHERSON, Dr. A. LASKIEWICZ, Mr. TERENCE CAWTHORNE, Mr. W. O. LODGE and Mr. HECTOR THOMAS; the opening speakers replied.

Section of Epidemiology and Preventive Medicine

President—MAURICE MITMAN, M.D., F.R.C.P., D.P.H.

[*October* 21, 1955]

Escherichia coli Serotypes in a Nursery

By J. C. McDONALD, M.D., D.P.H., and RUTH E. CHARTER, B.Sc., Dip. Bact.

Epidemiological Research and Salmonella Reference Laboratories, Central Public Health Laboratory, London

THE investigation described was carried out in a private residential nursery in Middlesex between December 1951 and November 1952. An infant had been admitted to hospital with gastro-enteritis from the nursery on 6.12.51 and *Escherichia coli*, Type 0125 B15, was isolated from its stools. In an earlier investigation by Taylor and Charter (1952) evidence suggesting the possible pathogenicity of this serotype was found, and for this reason it was thought that a careful study of the children in the nursery from which the sick child had come, would be worth while. The matron and the doctor in charge of the nursery gave permission for us to make clinical and bacteriological observations which, because of the events which followed, were continued for almost a year. At our first visit on 14.12.51 there were 12 children present, mostly under 1 year of age, and the staff consisted of the matron and another State-registered nurse, 2 nursery assistants and 2 domestic assistants. Observations were made on 48 children in all, 37 of whom were admitted or readmitted during the investigation. The number of children in the nursery fluctuated between 7 and 18 and their age distribution is shown in Fig. 1.
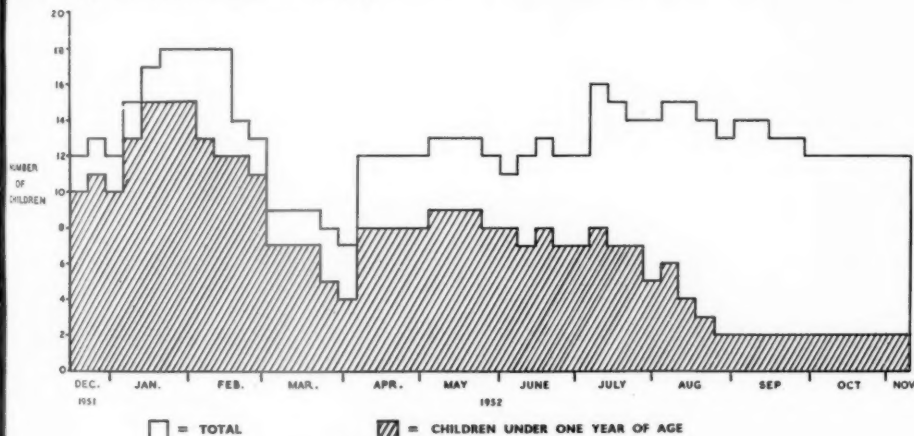
FIG. 1.—Age distribution of children in nursery during study.

Methods.—At a routine visit made by one of us at weekly intervals, the health of the children since the last visit was recorded and rectal swabs taken from them all. For three months, February to April, visits were made more frequently and swabs were taken two to three times a week during this period. The matron kept a daily stool record and weekly weight record for each child. A rectal swab was taken by the matron from all children as soon as possible after admission and immediately before discharge. The swabs used were dipped in broth before insertion in the rectum, and then taken to the laboratory where they were plated, usually within three hours, on blood agar, MacConkey agar and deoxycholate citrate medium. The blood and MacConkey plates were tested for the

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presence of serologically identifiable types of *E. coli* by the slide agglutination of 10 coliform colonies. All positive slide results were confirmed by tube agglutination. The detailed laboratory methods were similar to those described by Charter and Taylor (1952).

Results.—The main clinical and bacteriological findings are shown in Fig. 2. Five
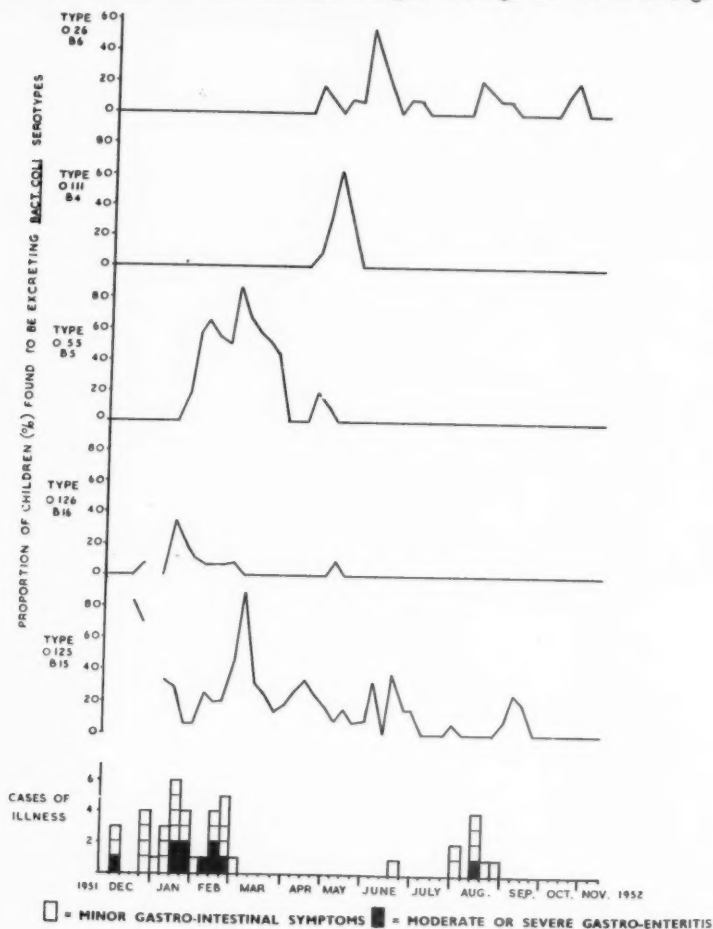
FIG. 2.—Bacteriological and clinical findings.

different serotypes of *E. coli* were isolated during the study. Type 0125 B15 was found in over 80% of the children at the first visit and remained in the nursery until September. Type 0126 B16 appeared at the end of December, Type 055 B5 in January and types 0111 B4 and 026 B6 in April. All 5 types were isolated from a considerable proportion of the children before disappearing from the nursery. In January and February there was a severe outbreak of gastro-intestinal illness; 9 children developed moderate or severe gastro-enteritis, and of 6 sent to hospital, 3 died. Minor gastro-intestinal symptoms were also frequently noted during the same period. In August a further case of gastro-enteritis occurred and others had minor symptoms.

The extent to which these illnesses were associated in time with the excretion of *E. coli* serotypes is shown in Table I. All 9 cases in the January–February outbreak occurred within seven days before or after the date on which *E. coli* Type 055 B5 was first isolated from them. 3 of the 9 cases were also found to be excreting Type 0125 B15 for the first time

TABLE I.—ASSOCIATION BETWEEN DATE OF FIRST ISOLATION OF AN *E. coli*
SEROTYPE AND ONSET OF GASTRO-INTESTINAL ILLNESS

E. coli serotype	Number of excreters		Number developing illness within a week before or after date of first isolation	
	Under 1 year	Over 1 year	Under 1 year	Over 1 year
026 B6	7	6	0	0
055 B5	18	5	9	0
			(3 fatal)	
0111 B4	8	3	0	0
0125 B15	18	7	3	0
			(also excreting Type 055 B5)	
0126 B16	11	2	0	0

within the same fourteen-day period. Illness was confined to those under 1 year of age but the average age of the 9 excreters of *E. coli* Type 055 B5 who developed gastro-enteritis was 3 months, whereas that of the 9 aged less than 1 year who remained healthy was 7 months. On the other hand 2 of the excreters were twins aged 9 months; the female remained symptom-free but the male became ill and died.

Bacteriological findings on the 9 cases associated with infection with *E. coli* Type 055 B5 are shown in detail in Fig. 3. It may be seen that not only was the onset of illness in every

■-TYPE O 55 B5   ▨-TYPE O 125 B15   ▦-TYPE O 126 B16

▨ - DURATION OF ILLNESS   SCALE ├─────┤ DAYS
                                      0   5   10
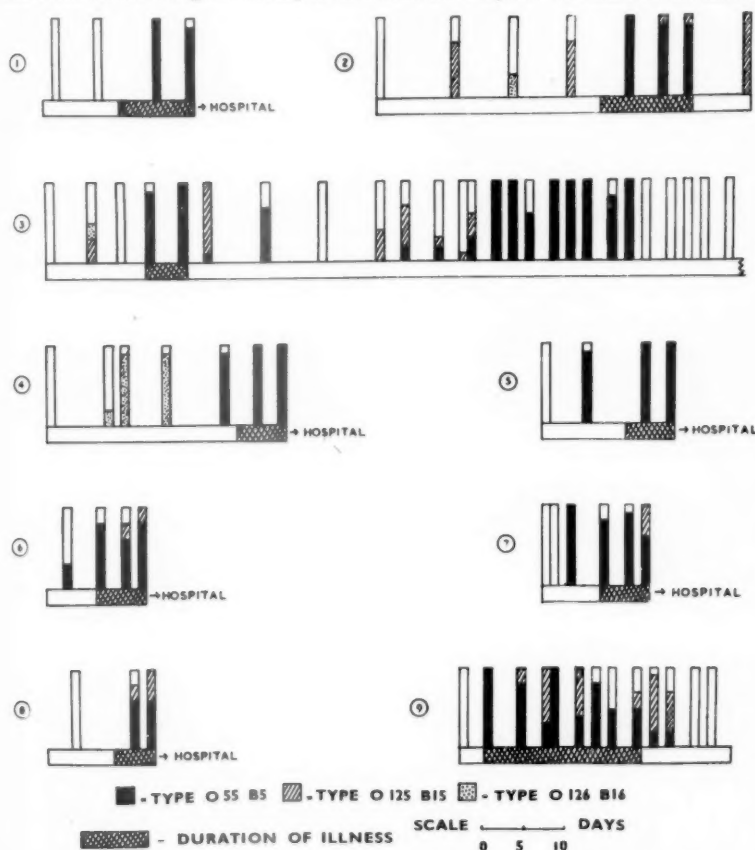
FIG. 3.—Bacteriological findings in illnesses associated with *E. coli* Type 055 B5.

Each vertical column indicates the result of the bacteriological examination of a rectal swab and shows the proportion and type of serologically identifiable colonies of *E. coli*.

case closely related to the first isolation of this serotype from a rectal swab, but also that the proportion of positive colonies on the plates was high during the period of illness. If it be concluded from this evidence that *E. coli* Type 055 B5 was the pathogen responsible for the epidemic, it is possible to give some indication of the approximate incubation period. In no case could it have been longer than ten days and the most frequently observed possible time was two to six days.

Infection with the other 3 serotypes found in the nursery—026 B6, 0111 B4 and 0126 B16—was not accompanied by any gastro-intestinal illness. The case of gastro-enteritis in August did not seem attributable to *E. coli* infection; Type 026 B6 was isolated from this child during the week before he became ill but no serologically identifiable strains were isolated during the illness. Although there is considerable past evidence of the pathogenicity of *E. coli* Type 0111 B4, carrier epidemics similar to the one we observed were also noted by Payne and Cook (1950) and Taylor and Charter (1952) among children in residential nurseries.

The manner in which the 5 serotypes were introduced into the nursery was not discovered. Of 37 children admitted during the study 26, swabbed within twenty-four hours of arrival, were negative, and 3 out of 11, swabbed at longer intervals after admission, were positive. *E. coli* Type 055 B5 was isolated from one child three days, and Type 0125 B15 from two children four days after admission, but both these types were already present in the nursery at the time. There was thus no evidence that any child introduced a new serotype into the nursery. Unfortunately no regular bacteriological examinations of the staff or of other visitors to the nursery were made and so it was not possible to test the suggestion made by Singer and Ludford (1953) that adult carriers may be responsible.

SUMMARY AND CONCLUSIONS

The natural history of *E. coli* infection in a group of young children was observed for almost a year. Five different serotypes were present at various times, each being isolated from a high proportion of children before disappearing from the nursery. A severe epidemic of gastro-enteritis occurred in the course of the investigation and provided strong confirmatory evidence for the pathogenicity of *E. coli* Type 055 B5. Some information on the probable incubation period was also obtained. Infection with the other 4 serotypes—Types 026 B6, 0111 B4, 0125 B15 and 0126 B16, was not accompanied by illness, though there is little doubt that the first two at least, have often been responsible for illness in other circumstances. As infection with potentially pathogenic serotypes of *E. coli* does not always result in illness, it is clear that other factors, at present unknown, play an important part in determining the outcome of such infections.

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Section of Obstetrics and Gynæcology

President—GERTRUDE DEARNLEY, M.D., F.R.C.O.G.

[*October* 28, 1955]

SPECIMENS were shown and case histories discussed as follows:

Intra-epidermal Carcinoma (Paget's Disease) of the Vulva.—MAGNUS HAINES, M.D.

Mrs. A. G., aged 65.

History.—Menopause fourteen years ago followed hysterectomy for uterine fibroids.

On examination (May 1955) for the complaints of a constant vaginal discharge since operation and irritation of the vulva for the past twelve months, there was acute vulvitis with copious exudate (stained smears showed monilia). On the left side of the vulva there was a purplish red raised area measuring 7 × 3 cm. which occupied almost the whole extent of the labium majus with slight involvement of the labium minus (Fig. 1). On the

FIG. 1.—Vulva. 5/7th natural size.
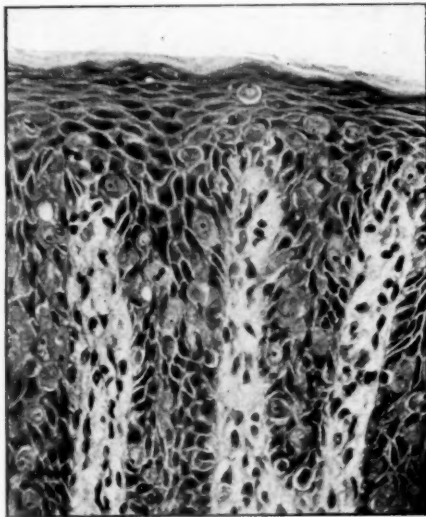
FIG. 2.—Paget cells in the layers of the epidermis. × 180.

right labium majus but not raised above the skin there were two smooth red glazed patches of from 2 to 3·5 cm. in diameter. No enlarged inguinal lymph nodes were felt. Biopsy and simple vulvectomy were performed. No recurrence up to five months after the operation (Mr. R. B. K. Rickford, Chelsea Hospital for Women).

Histology.—The findings at biopsy and in the excised vulva were similar and the changes seen in the left labial area were the same as those in the patches on the right. The layers of the epidermis showed no distortion of the normal pattern. In some places the rete pegs were elongated and in others they were stunted or fused. Every level was permeated by large vesicular epithelial cells of the Paget type (Fig. 2). The cells were fairly uniform in size, contained numerous mitotic figures and showed no resemblance to prickle cells. These cells were seen in the epithelium of some hair follicles but no evidence was obtained to show any connexion with sweat glands. The skin at the edge of the lesion did not show leukoplakia. Several blocks were made and many sections were examined. Serial sections of the whole specimen would be required before the presence of a small sweat gland carcinoma could be excluded.

Comment.—This is a case of intra-epidermal carcinoma of the vulva and judged on the histological findings it is an example of extra-mammary Paget's disease.

Different histological forms of intra-epidermal carcinoma have been described in the vulva but Willis (1953), amongst others, declares that they are not separate entities. On the other hand some writers (Plachta and Speer, 1954; Eisenberg and Theuerkauf, 1955) seek to demonstrate that the cells of Paget's disease are derived from neoplasms arising in the apocrine glands. Paget *et al.* (1954) described a case of Paget's disease of the left labium majus with an underlying adenocarcinoma and with Lennox and Pearse (1954) have attempted to reinforce their thesis by staining sections by periodic acid Schiff, the PAS method. A positive reaction, they state, indicates that the cells are not derived from the epidermis but by virtue of their positive staining reaction must belong to the epithelium of the glands. Some areas in the sections of the case now reported were PAS-positive but there is no evidence of a tumour of the skin glands.

Paget's disease of the vulva is rare and the problems of its precise ætiology and its relation to carcinoma of the vulva must await the results of further investigation of a greater number of cases.

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Self-induced Abortion Complicated by Gangrene of the Uterus and Anuria.—J. P. ROUX, M.B., Ch.B., M.R.C.O.G.

A 35-year-old woman, with two children aged 11 and 8, was pregnant for the third time. Her husband was on the point of leaving her, and at about the 20th week she took abortifacient tablets and douched herself with a blue liquid she had obtained. She fainted and commenced bleeding vaginally, losing half to one litre of blood.

Shock was successfully treated by the flying squad and she was transferred to hospital, blood being administered at the time.

The uterus reached to the umbilicus. There was marked tenderness and rigidity in the hypogastrium. No contractions were observed. The vulva and vagina were very œdematous. A cervical swab for aerobic and anaerobic culture was taken. Bleeding had ceased and the cervix was closed.

Fifteen hours after the administration of the douche, the patient again became shocked. Slight jaundice was noticeable, no urine had been passed. Serum taken was severely hæmolysed.

A Pitocin drip failed to induce contractions, her blood pressure fell to 80/50 and she developed severe air hunger, little relieved by oxygen. The diagnosis was made of shock, intravascular hæmolysis and pelvic peritoneal irritation due to self-induced abortion with a presumably caustic agent, and with the possibility of *Cl. welchii* infection. The patient was anæsthetized for abdominal evacuation of the uterus.

There was no incisional bleeding and the recti were an unhealthy purplish-brown colour. A swab was taken of a small amount of blood-stained intraperitoneal fluid. The uterus, ovaries, tubes, broad ligaments and bladder were dark brown in colour, and at hysterotomy it was obvious from the gangrenous state of the completely avascular myometrium that total hysterectomy was imperative. This was rapidly carried out and the appendages removed at the same time. The uterine and ovarian veins were seen to be thrombosed. Owing to the extent of gangrene, surgery had to be radical, the bladder being the only organ of suspect viability conserved.

Two hours after the operation, the patient's condition was much improved. She was warm, breathing easily, her blood pressure 130/90. After this her general condition remained satisfactory and she was comfortable and cheerful, but it was soon obvious that, as expected, anuria was going to be the ultimate problem.

No clostridia or other specific organisms were cultured from the swabs. Penicillin was, however, administered daily in dosage of one million units.

Bladder drainage was continued in view of the suspect viability of the bladder. The catheter was removed on the third day.

A further 550 ml. blood transfusion was given on the first post-operative day when her hæmoglobin was reported as 10 grams per 100 ml.

After this, the patient was on a strict peanut-oil/glucose regime (Bull *et. al.*, 1949). It was found, as in other cases supervised by Taylor (1955) of the Biochemistry Department at the

Radcliffe Infirmary, that an intermittent drip (on for two hours, off for two hours) was better tolerated. It was also found that the optimum daily intake was 800–900 ml. The current view (Bull, 1955) is that 800 ml. or even slightly less, are sufficient. Bull has also reduced his carbohydrate to an optimum of between 300–400 grams per day according to what the patient can tolerate, and has progressively reduced the fat-content of the mixture and sometimes omitted it altogether, as it may cause diarrhœa. He is certain that a high carbohydrate diet is sufficient.

For ten days, $\pm\frac{1}{2}$ to 1 oz. (15–30 ml.) of fluid per day was passed. It was not possible to decide whether this was urine or transudate.

On the eleventh day, 4 oz. (120 ml.), and on the twelfth day, 15 oz. (450 ml.), of urine were passed. After this, the amount increased day by day until within ten days more than 100 oz. (3 litres) were passed daily (Fig. 1).
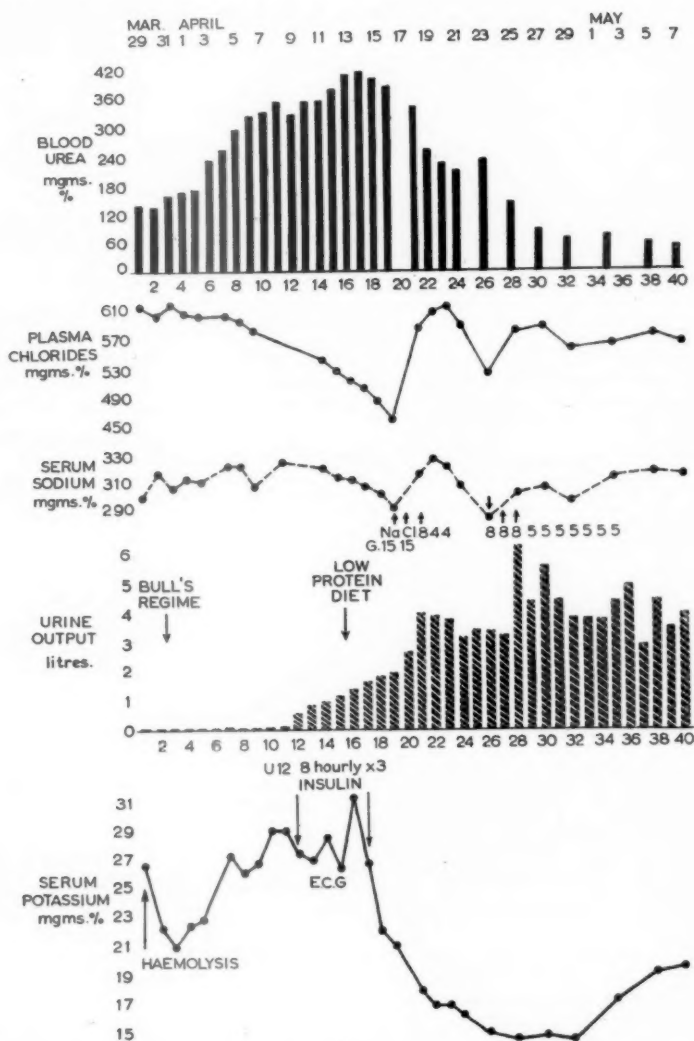

FIG. 1.—Graphic illustration of biochemical changes during anuric and diuretic phases.

The output was carefully measured in these first days of diuresis and an equal amount added to the intake (½ as water, ½ as normal saline). On the fourth day after resumption of excretion, when the output exceeded one litre, the peanut-oil/glucose mixture was replaced by a low protein diet (2,000 calories, 30 grams protein). This might have been too early, as the blood urea rose again, though it had been levelling out at 350 mg. per 100 ml. as diuresis commenced. The highest level reached was 414 mg. per 100 ml. on the seventeenth day.

When the serum potassium exceeded 29 mg. per 100 ml. on the tenth and eleventh days, and again, 31·4 mg. per 100 ml. on the sixteenth day, 12 units of soluble insulin were given eight hourly for twenty-four hours and on both occasions it dropped to below the danger level.

Electrocardiographic tracings taken on the fourteenth day, showed markedly amplified T-waves consistent with hyperpotassæmia.

Also illustrated is the well-marked fall in serum sodium and plasma chlorides in the diuretic phase, even though we thought we were giving enough salt by giving half the amount put out back as normal saline. The low values of 454 mg. per 100 ml. (plasma chloride) and 291 mg. per 100 ml. (serum sodium) were obtained on the nineteenth day, a week after excretion had recommenced. This was remedied by the administration of extra salt as shown, and again when a subsequent drop occurred a week later.

Penicillin was given on alternate days after the first ten days and discontinued a week later. Toxic agents such as Aureomycin and Terramycin would obviously do harm by cumulative effect in cases of anuria.

The abdominal and vaginal vault scars healed by first intention.

The story has a happy ending. The patient's husband hardly left the hospital premises during these weeks of crisis and a very happy couple went home on her forty-third post-operative day. She has been followed up twice since, has only occasional hot flushes, her blood pressure was 120/80, blood urea 30 mg. per 100 ml., her urine contained no albumin and had a specific gravity of 1020.

Summary.—A case of self-induced abortion complicated by gangrene of the uterus and anuria is presented. Treatment was by hysterectomy and the regime advocated by Bull and his co-workers. The patient made a satisfactory recovery.

To summarize the practical lessons learnt, they are:

In cases of anuria an intermittent gastric drip of the peanut-oil/glucose mixture is better tolerated; ± 800 ml. per day is sufficient during the anuric phase.

The mixture should not be discontinued too early, to be replaced by a diet containing protein, as the blood urea may continue to rise even after diuresis has commenced.

The importance of an adequate salt intake during the early diuretic phase is stressed.

The danger of a high serum potassium in the anuric and again in the early diuretic phase is recognized and the use of insulin in controlling this has been demonstrated.

The difficulty in differential diagnosis in cases of gangrene of the uterus following self-induced abortion rests mainly with the presence or not of *Cl. welchii* infection. In cases of doubt, the administration of penicillin is safe, but in view of the possibility of anuria, the prophylactic use of toxic antibiotics such as streptomycin, Aureomycin and Terramycin, which by cumulative effect would be dangerous, is contraindicated.

I am indebted to Mr. J. A. Stallworthy for permission to publish this report.

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Criminal Abortion with Gangrene of Uterus and Fallopian Tube.—J. S. MACVINE, F.R.C.S.Ed.

Mrs. F., aged 41. 3 children. 2 previous abortions.

Last menstrual period 10.4.55.

Admitted Central Middlesex Hospital on 21.7.55. History of having been syringed with soap solution. Aborted soon after admission and retained products of conception removed from cervical canal with ovum forceps in ward. Temperature subsided till 24.7.55. Clinically a general peritonitis and patient desperately ill. Laparotomy with view to possible drainage.

Findings.—General peritonitis with free blood-stained fluid in peritoneal cavity. Patch of gangrene 2 in. in diameter, right side of uterus. Complete gangrene right fallopian tube. Abscess right side of pelvis near base of broad ligament.

Procedure.—Very rapid subtotal hysterectomy and right salpingectomy. Free bleeding from abscess site controlled by plugging which had to be left in situ and was removed forty-eight hours after operation.
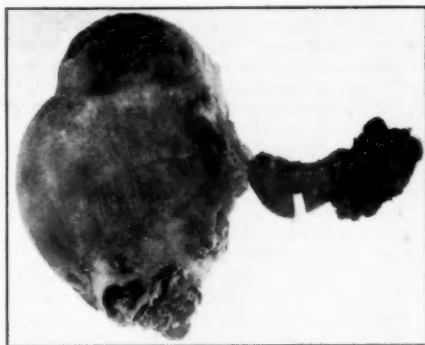
FIG. 1.   FIG. 2.

FIGS. 1 and 2.—Specimen shows a uterus and right fallopian tube which has been cut across to expose the uterine cavity. There is a circumscribed area of gangrene in the right side of the fundus. This was quite black at operation. The fallopian tube is also gangrenous. In the cavity of the uterus can still be seen retained products of conception.

Convalescence.—Stormy—paralytic ileus and bronchopneumonia. Temperature finally subsided by the twelfth day after operation.

24.8.55: Discharged to convalescent home after thirty-four days in hospital.

20.9.55: Seen in Outpatients and discharged.

Inversion of Uterus Due to Neoplasm.—C. W. TAYLOR, M.B., F.R.C.O.G.

A. F., female, aged 15, was admitted to the Birmingham and Midland Hospital for Women, under the care of Mr. P. J. Ganner, on 31.1.55. There was nothing of note in her past or family history. Her periods commenced at the age of 13 and were regular, cycle 7/28.

Present history.—Irregular vaginal hæmorrhage for ten months. Lower abdominal pain for three months.

Vaginal examination under anæsthesia showed a large fungating mass, ? extruding through the cervix. Biopsy was performed and showed a connective tissue tumour of indefinite type—the picture being obscured by necrosis.

At operation (8.2.55) vaginal examination, which was difficult, suggested that the tumour mass was arising from the fundus of an inverted uterus. The abdomen was opened and complete uterine inversion confirmed. The uterus and neoplasm were removed but the appendages, which were astride the rim of depression, were conserved. No pelvic or intra-abdominal deposits of growth were seen.

The patient was readmitted ten weeks later with a large abdomino-pelvic mass. Deep-ray therapy was commenced but the patient's condition deteriorated, uræmia ensued and she died on 6.5.55. Autopsy was refused.

Operation specimen.—Large polypoidal masses are seen arising from the fundus and lateral aspect of the body of the completely inverted uterus. The endometrium covering the rest of the uterus and cervix is smooth and congested. Histological examination shows a "sarcomatous type" of neoplasm with areas of myxomatous tissue containing clusters of embryonic cells. No areas of cartilage or bone are found and no cross-striations can be demonstrated. The appearances otherwise, however, are typical of mixed mesodermal tumour.

Intra-uterine Fœtal Death with Defective Maternal Blood-Clotting Mechanism.—BERYL O. HOWIE, M.B., Ch.B., M.R.C.O.G.

This case presents the problem of intra-uterine fœtal death complicated by serious defects in the maternal blood-clotting mechanism.

The patient was a Rhesus-negative multigravida with a most unfortunate obstetrical history.

1947: Full-term spontaneous delivery of a daughter weighing 6 lb. 9½ oz.—the only surviving child.

1950: Premature delivery at 36 weeks of a 5 lb. child who died forty-eight hours after birth from atelectasis and prematurity.

1951: 16 weeks miscarriage.

1952: Precipitate delivery at term of a 5 lb. 10 oz. infant who died two hours after birth from intracranial hæmorrhage. At post-mortem, also signs of hæmolytic disease—enlarged spleen and generalized œdema. Rhesus antibodies had been present in the maternal serum during the pregnancy.

1953: Intra-uterine death at 26 weeks due to hydrops fœtalis. Macerated stillbirth at 28 weeks. Antibodies again present.

1954: 16 weeks miscarriage. 20 weeks miscarriage.

i.e. only one living child from 7 pregnancies.

The patient was then remarried to a heterozygous Rhesus-positive husband. In the pregnancy under consideration—the first of the new marriage—Rhesus antibodies were again present in the early weeks but all was otherwise proceeding normally until 29 weeks when intra-uterine death again occurred.

At 33 weeks, four weeks after the intra-uterine death, the patient was admitted to the Horton General Hospital, Banbury, complaining of spontaneous bruising of one week's duration, with prominent bruises on the left forearm, right knee, and a very large extravasation of the left hip. No other abnormal bleeding had occurred. There was no previous history or family history of blood dyscrasia.

Blood investigations were commenced:

Hæmoglobin 89%; platelet count 123,000/c.mm.; one-stage prothrombin time: patient 45 sec., control 20 sec. (prothrombin ratio 2·3), i.e. the platelet count was rather low and the prothrombin time noticeably delayed.

The patient was transferred to the Radcliffe Infirmary, Oxford, the following day with vaginal bleeding.

Before the patient arrived word had been received from Banbury that after three-quarters of an hour no clotting had been observed in the sample of blood taken. A supply of fibrinogen was therefore obtained and was on hand when the patient arrived.

On admission.—History of painless vaginal bleeding for four hours. There was no shock, the pulse rate being 80 per minute and the blood pressure 115/80.

On examination the bruising was found as described. The fundal height corresponded to a 24 weeks' cyesis although the patient was at 33 weeks by her dates. The fœtal heart was not heard. The uterus was not contracting. A further sample of blood taken on admission also failed to clot.

One hour after admission uterine contractions began and with each there was an increasing vaginal loss. Spontaneous bleeding from the gums was also noted.

A blood transfusion with compatible blood was started, and fibrinogen solution (1·9 grams to 1 pint normal saline) into a second vein.

Two and a half hours after admission the fœtus with intact sac, complete with placenta was delivered rapidly and spontaneously with a sudden loss of a further two pints of incoagulable blood. Transfusions of blood and fibrinogen solution were given more rapidly. Fortunately the uterus remained well contracted.

Three hours after admission—half an hour after delivery, blood taken clotted in ten minutes but rapidly redissolved.

Two hours after delivery, blood clotted in five minutes, the clot being stable and normal for the first time. Spontaneous bleeding from the gums ceased.

The total loss, estimated at 5 pints, was replaced by 4 pints compatible blood and two pints normal saline, each containing 1·9 grams fibrinogen.

The patient's subsequent progress was uneventful. She was discharged to Banbury on the tenth day. (Hæmoglobin 75%.)

Fibrinogen.—There was no fibrinogen present on admission, and the plasma fibrinogen level was still low, though not below the critical level, three days after delivery (Table I).

TABLE I.—PLASMA FIBRINOGEN LEVELS

Time	Plasma Fibrinogen [mg.%]
On admission ..	Nil
3rd day after delivery	175
6th ,, ,, ,,	295
6 weeks ,, ,,	310

Normal values for pregnant subjects = 315 mg.%
  ,, ,, (non-pregnant) ,, = 267 mg.%
  ,, ,, level, below which no clotting occurs: 90 mg.%
(Hodgkinson *et al.*, 1955).

Platelets.—The platelet count was low prior to delivery, there were no platelets present on admission, and again abnormally low counts for several days after delivery (Fig. 1).
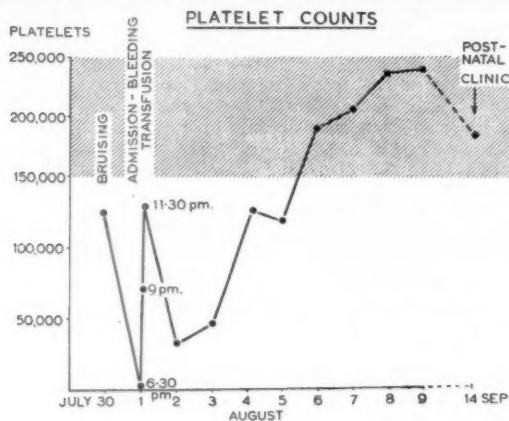

FIG. 1.

Other clotting factors.—Although the various factors were unfortunately not able to be defined qualitatively at the time, it was clear that there was also a deficiency of some of the other associated factors which normally play a part in the blood-clotting mechanism, i.e. prothrombin and Factors V and VII. One or more of these constituents were present in abnormally low amounts both before and after delivery as indicated by the unduly prolonged one-stage prothrombin time (Table II). (Where the prothrombin time values were low the tests were rechecked using blood to which fibrinogen had been added.)

TABLE II.—ONE-STAGE PROTHROMBIN TIMES

	Control	Patient	Prothrombin ratio
Before delivery ..	20	45	2·3
1st day after delivery	14	18	1·3
9th ,, ,, ,,	16	16	1·0

The absence of fibrinogen, or of platelets, or the deficient amounts of the associated clotting factors could each separately have caused a serious defect in the blood-clotting mechanism, but together led to drastic failure of normal clotting.

Discussion.—Defective blood clotting associated with pregnancy and delivery has only recently been recognized as an entity. It may have been the underlying cause of maternal deaths from post-partum hæmorrhage previously thought to be associated with an "irreversible" stage of shock.

Recent writers (Weiner *et al.*, 1950*a*, *b*; Schneider, 1955; Steiner and Lushbaugh, 1941; Dieckmann, 1936; Barnett and Cussen, 1954; Hodgkinson *et al.*, 1955; Frick and McKelvey, 1955) have reported that it is likely to occur in cases of (1) Abruptio placentæ. (2) Intrauterine death with retention of the fœtus *in utero*. (3) Amniotic fluid embolism.

In each of the above instances the causative mechanism is similar. The clotting defect was previously believed to be due to afibrinogenæmia, but the present case shows that other factors may be involved.

Amniotic fluid or particles of placental tissue, both rich in thromboplastin, gain access to the maternal blood stream where the thromboplastin initiates a process of extensive intravascular clotting. This takes fibrinogen out of solution faster than its replacement, and defibrinates the blood. At the same time platelets are entrapped in the fibrin meshwork and are also removed from the circulation, again even faster than their usual rapid replacement. The minute clots or fibrin emboli are rarely seen at post-mortem, either because they are rapidly dissolved, as they are formed, by circulating fibrinolysins, or alternatively because it is believed that the fibrin is laid down along the lining of blood vessels.

Violent uterine action accelerates the passage of liquor or placental fragments into the maternal blood stream, especially where membranes are ruptured or when uterine sinuses are laid bare as in abruptio placentæ. The use of Pitocin is contraindicated in cases of

intra-uterine death and accidental antepartum hæmorrhage for this reason, because of the risk of its inducing sudden powerful contractions.

If the thromboplastin-rich substance enters the blood stream very suddenly as in massive amniotic embolism, the sudden extensive intravascular clotting which occurs causes an anaphylactoid reaction, with dyspnœa, cyanosis and possibly sudden death. Occasionally chest X-rays taken during the acute phase show an appearance like that of miliary tuberculosis due to the presence of multiple tiny infarcts, possibly caused by fibrin emboli—but these appearances are usually fleeting only. If the patient survives the acute attack gross clotting defects are likely to complicate delivery.

With intra-uterine death, the onset of symptoms such as bruising and spontaneous bleeding is insidious due to the gradual chronic absorption of liquor—the "dead fœtus syndrome" described by Weiner and his co-workers (1950a, b.)

Early writers especially emphasized the importance of cases where the intra-uterine death was due to Rhesus incompatibility, but there is no statistical proof that clotting defects are any more common in this group than in cases of intra-uterine death from other causes.

Practical Application in Clinical Management.

(1) *Early diagnosis* is important so that adequate preparations can be made (e.g. fresh blood and fibrinogen obtained) and the emergency adequately dealt with early in the appropriate manner before the situation becomes uncontrollable.

(2) *Investigation of suspected cases.*—(a) *Weiner's clot observation test.* (5–10 c.c. blood into plain sterile tube left to stand and observed.) The clotting time and the stability of the clot are proportional to the amount of fibrinogen present. If clotting is delayed for more than ten minutes or if the clot dissolves within one hour there is a serious deficiency.

If the fibrinogen level is 0–60 mg.%, no clotting occurs.

    60–100 mg.%—soft clots only, rapid dissolution.
    100–150 mg.%—partial dissolution.
    150 mg.% and over—normal clotting.

(b) *Platelet count*—to determine any associated platelet deficiency.

(c) *One-stage prothrombin time*—to detect deficiency of associated factors.

(d) *Chest X-ray*—When respiratory symptoms present, very occasionally a typical picture of the fleeting multiple pulmonary infarcts is obtained.

(3) *Treatment*—(a) *Fibrinogen* is given intravenously and rapidly, usually about 4 grams in the first instance, the total amount being dependent on its effect in producing more normal clotting at serial clot observation tests. Fibrinogen, however, is not sufficient alone.

(b) *Blood* is required in addition. Stored blood, contrary to popular belief, does, in fact, contain platelets in considerable numbers though some doubt has been cast on their efficacy in clot formation. Fresh blood makes good any platelet deficiency, but it also is the *only* means of supplying the other factors required for normal clotting. Because of the possible deficiency of these factors blood loss should be replaced fully with fresh blood if available.

(c) *Calcium.*—Finally, if renal function is impaired as is often the case in concealed accidental antepartum hæmorrhage, and if much blood is given by transfusion, one may find defective clotting because of retention of the citrate given with the blood for transfusion. (This is normally excreted by the kidneys very rapidly and usually does not constitute a problem.) In these few cases it may be necessary to administer calcium in the form of calcium gluconate for example (10 c.c. of a 10% solution, given very slowly by intravenous injection).

Conclusion.—The case presented is that of intra-uterine death, retention of the fœtus *in utero* and the insidious onset of defects in the clotting mechanism causing firstly spontaneous bruising, then excessive bleeding at the time of delivery together with a sudden exacerbation at that time leading to respiratory distress.

It illustrates the various factors which can lead to defective clotting, the importance of early diagnosis, the investigations which can lead to the establishment of the diagnosis, and indicates the treatment required to deal with the several specific deficiencies which may be present.

I am indebted to Mr. J. A. Stallworthy and Mr. W. Hawksworth for permission to present this case report.

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Section of Radiology

President—E. Rohan Williams, M.D., F.R.C.P., F.F.R., D.M.R.E.

[*October* 21, 1955]

DISCUSSION ON THE POSSIBLE SIGNIFICANCE OF THE THYMIC ORIGIN OF HODGKIN'S DISEASE

Dr. A. D. Thomson (Bland-Sutton Institute of Pathology, Middlesex Hospital):

In opening this discussion on the concept of the thymic origin of Hodgkin's disease and its possible significance to both pathologists and clinicians, I hesitate to add yet one more histogenetic possibility to the large number of diverging opinions that already exist as to the precise nature of Hodgkin's disease. By doing so, I hope that certain practical points may emerge for the further elucidation of this puzzling disease.

In a recent publication (Thomson, 1955) it was suggested that Hodgkin's disease was a tumour arising in the thymus gland and metastasizing from this site to give the clinical manifestations of Hodgkin's disease.

The detailed histological pattern of Hodgkin's tissue with its mononuclear cells, "mirror image" and "Dorothy Reed" giant cells, lymphocytes and eosinophils incorporated within a fibrous stroma, has been firmly established since the beginning of the present century. While these histological characteristics have ceased to be controversial issues, the precise nature of this polymorphic cellular pattern remains in doubt.

Many workers have regarded Hodgkin's disease as an infective granuloma and a large variety of organisms, including tubercle bacilli, viruses, diphtheroids, brucella organisms and parasites have been isolated from the affected tissues and claimed by their several founders as the causative agents. None of these bacterial claims has been fully substantiated and it appears that each of these incriminated organisms was merely a secondary invader in already diseased tissues.

By exclusion of this inflammatory ætiology a neoplastic nature becomes the more probable, but as Anderson (1953) remarks: "It is difficult to conceive a neoplasm in which the tumour cells are of so many different types."

The most favoured method of circumnavigating this polycellular histogenetic problem is to invoke the reticulum cell, with its diverse powers for differentiation, as the cell of origin and to state "There is now no room for doubt that Hodgkin's disease is a form of sarcoma of lymphoid tissue, derived from the bipotential stem cells, and exhibiting differentiation predominantly of reticular cell type with accompanying fibrosis, but also to a variable degree of lymphoid type" (Willis, 1948).

Before accepting this dogmatic statement it will be recalled that Hodgkin's disease has been known to arise from the tissues of the thymus gland and is classified as one type of thymic tumour (Symmers, 1933).

The thymus gland has a complex histological structure as it contains both lymphocytes and epithelium. The epithelium is represented by the multinucleated Hassall's corpuscles. During their normal development these structures originate from a large mononuclear epithelial cell which then divides to form an "owl's eye" or "mirror image" type of giant cell. Further progression results in cells containing from 3 to 20 or more nuclei until the fully developed Hassall's corpuscles are formed. Eosinophil polymorphonuclear leucocytes are also present in normal thymic tissue.

If these thymic mononuclear cells, "mirror image" giant cells and multinucleate giant cells are incorporated in a fibrous stroma with an admixture of lymphocytes and polymorphonuclear leucocytes the histological picture of Hodgkin's tissue is produced.

Case I (a patient of Dr. Frances Gardner's) was that of a young man who was found to have a mass in the anterior superior mediastinum on a routine chest X-ray. A thymectomy was performed (Mr. G. Qvist) and the histology of this specimen (Fig. 1) revealed the picture of Hodgkin's disease. There was no evidence of spread of this tumour beyond the confines of the mediastinum.
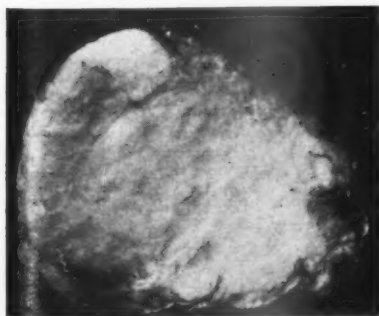
Fig. 1 (*Case I*).—Thymectomy specimen. (Actual size = 3½ in. across.)

Case II was that of a woman aged 35 who presented with enlarged glands in the left cervical region. These were removed by block dissection and the histology showed Hodgkin's tissue. A thymectomy was later performed (Mr. D. H. Patey) and the thymic tumour was removed together with enlarged lymph nodes extending upwards towards the left neck (Fig. 2). The histology showed Hodgkin's disease of the thymus gland with replacement of the lymph nodes by similar tissue.

This case illustrates an example of Hodgkin's disease originating in the thymus with metastatic involvement of the mediastinal and cervical lymph nodes.

FIG. 2 (*Case II*).—Thymectomy specimen with invasion of lymph nodes.

FIG. 3 (*Case III*).—Thymectomy specimen with a nodule of tumour in the upper part of the right lobe.
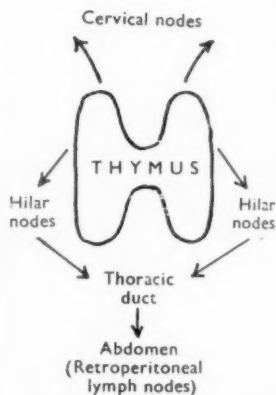
FIG. 4.—Diagrammatic representation of the lymphatic drainage of the thymus gland.

FIG. 5 (*Case IV*).—Thymic tumour with hilar lymph node invasion.

FIG. 6 (*Case IV*).—Showing involvement of the sternum with invasion of the internal mammary chain.

Case III (a patient of Sir Daniel Davies') was a female aged 25, who presented with enlarged glands in the neck. Biopsy of one of these showed replacement by Hodgkin's tissue. A chest X-ray at this time revealed an upper anterior mediastinal mass extending to the right of the sternum. The patient was treated by irradiation to the neck and mediastinum to a tumour dose of 2,000 r, with disappearance of the mediastinal shadow as seen on the chest X-ray.

Subsequently a thymectomy was performed (Mr. D. H. Patey) and the specimen (Fig. 3) showed a thymus of normal contour but with a firm focus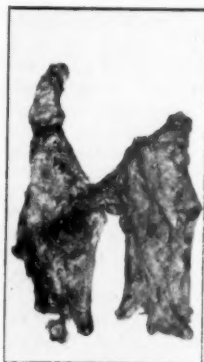 in the upper part of the right lobe. Histological examination of this area revealed the presence of viable tumour cells within the thymus gland.

This case illustrates Hodgkin's disease of the thymus with metastases in the cervical lymph nodes and the presence of viable tumour in the thymus following irradiation.

If the thymus is postulated as the primary site of Hodgkin's disease how are the metastatic manifestations of this disease produced? Fig. 4 is a diagrammatic representation of the normal lymphatic drainage of the thymus gland. It can be seen that there are lymphatic paths extending upwards to the cervical nodes, posteriorly to the tracheobronchial group and hence into the thoracic duct and by retrograde spread to the retroperitoneal lymph nodes of the abdomen. There is also lymphatic drainage anteriorly to the internal mammary chain. Cases II and III have already demonstrated the cervical route of dissemination.

Case IV was a man aged 30 with Hodgkin's disease diagnosed on biopsy of a cervical lymph node. He was treated by irradiation but the disease progressed rapidly and he died.

The autopsy revealed extensive mediastinal involvement with a thymic tumour and hilar node invasion (Fig. 5), internal mammary chain invasion (Fig. 6), and involvement of the spleen (Fig. 7).

FIG. 7 (*Case IV*).—Nodules of Hodgkin's tissue in the spleen.

FIG. 8 (*Case V*).—Thymic tumour in the mediastinum.

Case V was that of a man aged 35 with Hodgkin's disease who was treated by irradiation but the disease was generalized at the time of diagnosis and he died six months later.

Autopsy showed a thymic mass (Fig. 8) with extensive invasion of the abdominal nodes and kidney (Fig. 9) and a typical Hodgkin's pattern in the enlarged spleen (Fig. 10).

Cases IV and V demonstrate, therefore, Hodgkin's disease having thymic tumours and showing evidence of widespread dissemination of the disease to involve the hilar nodes, the internal mammary chain, the retroperitoneal tissues and the spleen.

These five recent examples of Hodgkin's disease all had thymic tumours, but in other cases the primary lesion may not be so obvious. The possible reasons for the failure to find the thymic primary are a very small tumour, sclerosis of the lesion, or the tumour may arise from ectopic thymic tissue either in the neck or from an abnormal site within the chest.
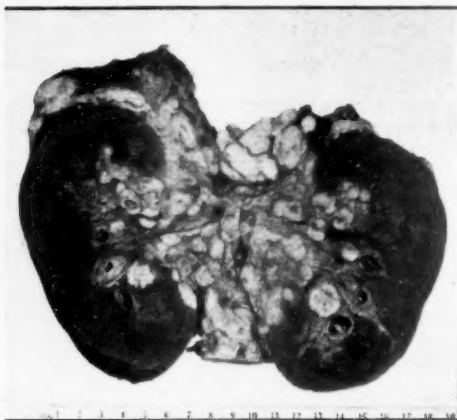
FIG. 9 (*Case V*).—Invasion of abdominal lymph nodes and the kidney.

FIG. 10 (*Case V*).—Showing the splenic involvement by Hodgkin's tissue.

It is certain that many of the cases diagnosed as Hodgkin's disease in the past are true examples of metastatic thymic tumour, and it is a pathological possibility that most, if not all, examples of Hodgkin's disease arise in the thymic gland or from ectopic thymic tissue.

If the concept of the thymic origin of Hodgkin's disease is accepted even in some cases, certain practical issues emerge from this work. Firstly, are not many of the mediastinal masses seen on the chest X-ray in fact thymic tumours rather than enlarged mediastinal lymph nodes? Secondly, is sufficient attention paid to the mediastinum by radiotherapists and in sufficient dosage? Thirdly, has thymectomy a place in the treatment of Hodgkin's disease either alone or in conjunction with radiotherapy?

My thanks are due to Mr. D. H. Patey for the thymectomy specimens of Cases II and III; to Mr. G. Qvist and Dr. A. G. Stansfeld for the specimen of Case I, and to Dr. W. W. Richardson and Dr. R. E. Cotton of the Bland-Sutton Institute of Pathology, Middlesex Hospital, for the autopsy specimens in Cases IV and V.

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Dr. A. M. Jelliffe (Meyerstein Institute of Radiotherapy, Middlesex Hospital):

As a student two facts about Hodgkin's disease became fixed in my mind: (1) That it is probably multifocal in origin, and (2) that it is inevitably fatal, usually at an early date.

Later I found that some authorities believed it might arise in one focus and from there progress systematically onwards, and that long survivals were possible. Dr. Thomson and I have reviewed all cases of Hodgkin's disease seen at the Middlesex Hospital since 1930 and the behaviour of the disease in many patients did suggest an orderly progression from a single site. In a few cases a long survival, without recurrence of disease, was found. The longest time recorded in this series was nineteen years, the patient still remaining free of disease. Such a happy outcome is unfortunately uncommon but if it is accepted that it can occur and that the disease may be unifocal in origin, then a more optimistic approach can be adopted.

The most important features influencing the progress of the disease appeared to be the histological appearances of the tumour, the age and sex of the patient and stage reached by the disease when the diagnosis is first established. We have staged our cases as is shown in Table I, believing that constitutional symptoms indicate that the disease is widespread. In this, and in other respects, our staging differs from that used by Peters (1950).

TABLE I.—STAGING OF HODGKIN'S DISEASE

Stage I. Lymph node involvement in only one main group.
Stage II. Lymph node involvement in 2 or more adjacent groups in either upper or lower half of body.
Stage III. (a) Generalized lymph node involvement.
  (b) Constitutional manifestations for which no other reasonable cause can be found.
  (c) Disease apparently limited to retroperitoneal lymph nodes.
  (d) Involvement of structures other than lymphatic.
(From *Brit. J. Cancer* (1955), **9**, 21.)

When Dr. Thomson first told me of his idea of the possible thymic origin of Hodgkin's disease, I was very sceptical. I was too well acclimatized to the reticulum cell theory of origin. Since then, my views have changed. If it does prove to be correct it could explain many of the peculiarities of the disease.

Recent American publications have described primary thymic tumours composed of tissue remarkably similar to Hodgkin's disease. In the Middlesex Hospital cases there were many with anterior mediastinal shadows closely resembling the American thymic tumour and other series have recorded the same finding. Jacox, Pierce and Hildreth (1936) show chest X-rays of 6 patients. 2 of these could be typical thymic tumours.

Hodgkin's disease rarely gives rise to upper mediastinal compression. In the Middlesex Hospital series (Jelliffe and Thomson, 1955), one out of a total of 227 patients showed this complication. A possible explanation could be that most of the shadow seen on the chest X-ray lies in the anterior mediastinum and does not immediately surround and compress the trachea and great vessels. Not infrequently, there is involvement of the sternum, anterior displacement of the thoracic cage and parasternal node involvement in Hodgkin's disease. This would fit in with the thymic concept. Goldman and Victor (1945) also found a high incidence of parasternal lymph node enlargement.

The most important idea that follows this new suggestion is that it may be possible to do more for the patient. Unfortunately, at least 50% of cases have generalized disease when the patient first presents himself. Radical treatment to the thymus cannot then hope to prolong life, and it may well influence the prognosis adversely. The only benefit possible following the renaming of the disease at this stage might be an improvement in the mental condition of the patient who knows he has that apparently invariably fatal condition, Hodgkin's disease.

If the disease is localized in extent, then radical treatment might be expected to improve the prognosis, if this new idea is correct. Dr. Thomson suggests that the disease may arise in the neck or mediastinum. It is likely that it will be in the latter site in at least four out of five cases, but presumably both sites would have to be treated. There are two possible methods by which a radical cure would be attempted.

Surgical excision has been advised in the past, and a few long term successes have been recorded (Table II). If Dr. Thomson's suggestion is correct, it might appear that surgery

TABLE II.—HODGKIN'S DISEASE: RADICAL RESECTION OF PERIPHERAL NODES

Authors	Date	Resections	Results
Wright	1938	1	1 case (C) A and W 11 years
		Excised twice X-rays also	
Baker and Mann ..	1940	5	1 case (C) A and W 10 years
		X-rays also	1 case (X) A and W 12 years
O'Brien	1941	1	1 case (C) A and W 19 years
			Rec. 20 years
Slaughter and Craver	1942	5	1 case (C) died 10 years
		X-rays also	(active)
			1 case (C) A and W 8 years
			1 case (C) A and W 11 years
			1 case (G) A and W 5 years
			Died 6 years
			Med. rec.
Jelliffe and Thomson	1955	5	1 case (C) survived 5 years
		X-rays also	

C = Cervical.   X = Axillary.   G = Inguinal.

may be of greater benefit in the future. The generally accepted tenets of cancer surgery are that the diseased organ should be removed with its fascial connexions and its lymphatic field, where practical, in one block. If this is accepted in this case, then the thymus should be removed in continuity with the cervical, internal mammary, anterior mediastinal and tracheobronchial groups of lymph nodes. Radical pneumonectomy, as advocated by Brock and Whytehead (1955), is clearly a reasonable proposition when dealing with carci-

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noma of the bronchus, a disease which has responded poorly to less radical forms of treatment and which is rarely curable when seen by the radiotherapist. I do not think the even more radical operation is justified in a disease which is extremely radiosensitive. The problem closely resembles that met with in seminoma of the testicle where irradiation to the abdomen gives the best results. Unsuccessful attempts at radical removal will lead to scarring and a reduction in blood supply with a decrease in the radiosensitivity of the tumour. It should be considered only when irradiation has failed.

Assuming Dr. Thomson's views are correct, radiotherapy would offer a more logical approach to the problem, with a real hope of cure in probably not more than 20% of all cases. When the disease is apparently limited to the neck, or to the neck and thorax, a radical course of X-rays should be given to both regions. There is little available information as to the quantity of radiation necessary to ensure inactivation of a diseased area in Hodgkin's disease. From our review of the Middlesex series we believe that at least 3,000 r in three to four weeks is necessary if there is to be a good chance of permanent regression in the treated area. In most cases this dose level can be achieved without undue difficulty. If conventional deep X-ray therapy appears unlikely to achieve this dose, as will be the case if the patient is thickest, it may be necessary to use grid therapy or supervoltage irradiation.

Prophylactic irradiation has been advocated in the past. Peters recommends treatment to all the unaffected lymph node groups of the body. If the disease is indeed multifocal in origin there is little hope that this will be a practical measure. If the disease originates in the anterior mediastinum or neck, prophylactic irradiation seems worth considering, although a dose level to that suggested of at least 3,000 r is not likely to be achieved over a large area.

Radical treatment to patients with already widespread disease not only appears useless, but may be harmful. The most careful selection of cases is necessary to exclude patients with generalized disease. The method of staging the Middlesex cases has been referred to previously. If there is any real doubt, it is advisable to place the case in the earlier of two stages. This will prevent an artificial improvement in the results, and will allow the patient the benefit of the doubt when consideration is being given to the best method of treatment.

There are several points which can be reasonably raised against this new idea. The *Lancet* (1955) recently published a well-balanced and constructive criticism of Dr. Thomson's work. I have two further criticisms of my own. The first is that I cannot fit into this new theory the rare case of mycosis fungoides that may continue for up to twenty years before the patient finally develops generalized Hodgkin's disease. The second is that occasional cases of Hodgkin's disease have been reported with the apparent site of origin far removed from the thymus or neck. The longer the survival of the patient without the appearance of disease in those regions, the less satisfactory the theory of the thymic origin of Hodgkin's disease becomes.

Twenty years ago Dr. W. M. Levitt said: "Suppose that it could be shown that in a substantial percentage of cases the disease began in, say, a certain group of abdominal glands, there would be at least a ray of hope that, if this group of glands were adequately irradiated, a permanent result might be obtained in at least a small proportion" (Levitt, 1934).

Dr. Thomson's work suggests that the word "thymus" may be substituted for "a certain group of glands". Autopsy findings are unlikely to be helpful, as invasion of the thymus at this stage would not necessarily indicate that the disease originated there, and absence of disease in the thymus might be explained by spontaneous regression of the disease, or by the almost universal use of the cytotoxic poisons when Hodgkin's disease is widespread.

I think this theory must stand or fall on the findings in the thymus at an early stage of the disease. The thymus can be easily removed after splitting the sternum and at the moment there is no reason to believe that this procedure leads to a more rapid dissemination of the disease. The patient suffers little discomfort and is usually eating normally the following day. It seems advisable to follow the operation by a full course of radiotherapy, with the shortest possible delay.

On the possible significance of the thymic origin of Hodgkin's disease, though the basic hypothesis is as yet unproven, I do feel that anything that might improve the prognosis in this tragic disease should be considered very carefully.

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Professor D. W. Smithers (Royal Marsden Hospital, and Institute of Cancer Research: Royal Cancer Hospital):

I think that Dr. Thomson and Dr. Jelliffe have made the case that Hodgkin's disease is commonly associated with thymic involvement (*see also* Thomson, 1955); I suspect that they are right in saying that it may—at least at times—arise primarily in the thymus. It has long seemed to me that the evidence that Hodgkin's disease often metastasizes is strong and it has been my practice to treat these patients with radical irradiation of each local manifestation of this disease as soon as it is detected and whether giving rise to symptoms or not. It does not yet seem clear to me, however, that they have either disproved the theory of the multicentric origin of Hodgkin's disease, or shown that it always arises primarily in the thymus, or that it is an entirely separate entity from reticulum cell sarcoma on the one hand and lymphatic leukæmia on the other.

The arguments in favour of the thymic origin of Hodgkin's disease seem to be of three kinds: (1) That all cell types are present in the normal fœtal thymus; (2) that there are similarities in age, clinical manifestations, sites of spread and histological appearances between thymic tumours and Hodgkin's disease; and (3) that thymectomy in Hodgkin's disease has shown the thymus to be involved and has demonstrated spread in a continuous chain of lymph nodes in the neck and parasternal regions.

The cell types are common and not inconsistent with the theory that the malignant elements may be derived from lymphocytes and reticulum cells. The similarities in behaviour with other thymic tumours do not seem impressive: thymic tumours have not been shown to metastasize to the spleen more readily than others, whereas Hodgkin's disease probably involves the spleen in 60–80% of cases. The striking predominance of renal metastases claimed for thymic tumours (Willis, 1952) is not matched in Hodgkin's disease. The evidence of thymectomy, however, though scanty is already impressive; the more operations showing thymic involvement, the better will the argument be, but a few negative findings in non-irradiated patients would reflect briskly against the theory.

The arguments in favour of Hodgkin's disease being associated with the reticuloses and leukæmias are not perhaps so sound that they cannot be shaken, but they still require consideration. The frequency of transitions or associations with reticulum cell sarcoma, lymphatic leukæmia and lymphosarcoma referred to by Willis (1953) and reported by Gill and McCall (1943) or Custer (1953), for example, must be accounted for or disposed of if Hodgkin's disease is to be established as a primary thymic carcinoma. I cannot judge the value of cytochemistry in this discussion, but the Ohio State University work (Ackerman *et al.*, 1951) producing evidence that the reticulum cell gives rise to the Hodgkin's cell may perhaps be of importance.

The relationship between the thymus and lymphoid tumours and their hormone control has long interested me. The following observations are given merely to indicate some of the things I have had in mind. The thymus was found to be the site of origin of virtually all radiation-induced lymphomas in strain C57 black mice following whole body irradiation, but not after local treatment over skull, mediastinum, lumbar region and tail (Kaplan, 1948, 1949), and thymic irradiation may produce leukæmia in man (Simpson *et al.*, 1955; Dameshek, 1954). Thymectomy prevents the development of radiation-induced lymphomas in mice (Kaplan, 1950). Adrenalectomy in rats leads to regeneration of retrogressed thymus in old animals and a stimulation of the thymus in young animals, inducing greater receptivity to transplanted leukæmia (Sturm and Murphy, 1944). Adrenal grafts, pituitary grafts and ACTH induce lymphoid tumours in mice (Silberberg and Silberberg, 1949, 1955; Silberberg *et al.*, 1953). Cushing's syndrome in man may be associated with thymic tumours and Thorne (1952) reviewed 10 cases from the literature of thymoma occurring in conjunction with this syndrome. Reports of interest in this connexion have come from Leyton *et al.* (1931), Kepler (1933) and Hubble (1949). The effect of thymolytic hormones (progesterone, testosterone, cortisone) on lymphoid tumours is said to be directly proportional to their effect on thymic weight (Kaplan, 1954). It seems that stimulants and irritants to the thymus and hormone changes increasing thymic weight promote the induction of lymphoid tumours, while thymectomy and thymolytic hormones prevent their occurrence or assist in their regression. In the lymphoid group of tumours, therefore, the thymus would seem to be of special interest both as one of the sites of origin of local tumours and as being associated with their induction elsewhere.

If the thymic origin of all cases of Hodgkin's disease is established so that it is seen invariably to be a metastasizing thymic carcinoma, then a pattern of preferential spread to lymphatic structures such as the spleen and lymph nodes—unlike that encountered with other carcinomas—must be assumed, and the association with other lymphoid tumours of multifocal origin be disproved. That Hodgkin's disease frequently involves the thymus and that it may originate there—perhaps frequently and possibly in every case—is a stimulating and interesting concept.

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Mr. T. Holmes Sellors stated that his contribution to the discussion was largely of "historical" nature. During the course of thoracic surgery he had encountered a number of anterior mediastinal tumours whose behaviour could not be classified under any known heading. There were tumours that were responsive to radiation but which, after excision, were reported as histologically benign; at the other extreme there were encapsuled tumours which had all the outward appearances of an innocent mass and yet were reported microscopically as lymphosarcoma or a tumour of highly malignant nature. When it was realized that a number of these were of thymic origin the picture became more clear, and a division into the thymocyte and epithelium forms helped to clarify matters, but there was a group which fitted into neither of these histological forms.

The usual appearance of these tumours suggested a granuloma which had a tendency to invade the innominate veins or superior vena cava, and from a surgical point of view was irremovable. These masses responded readily to radiotherapy and on many occasions have remained free from any sign of recurrence or symptoms over several years. It is this group that most resembles lymphadenoma, particularly when glands in the base of the neck and elsewhere become associated with the tumour at some stage of its career.

A point, however, that has proved puzzling has been the lack of association of this granulomatous form of tumour with myasthenia gravis. It is recognized that severe forms of myasthenia gravis may be associated with a thymic tumour as well as with an enlarged thymus. The tumour may be of benign or malignant character, but up to the present time he had not encountered any of the granulomatous types in which the patient had had signs or symptoms of myasthenia gravis.

At the present time he felt that tumours in the anterior mediastinum, if not possible of accurate diagnosis, should be treated by a full course of radiotherapy and then explored. In some instances there is no chance of excision, the tumour area being replaced by a matted, infiltrated mass which, though shrunk in size, is inextricably mixed up with mediastinal structures. On more fortunate occasions the tumour may be recognized as being part of the thymus and can be excised *en bloc*. On two occasions this has involved sacrificing the superior vena cava but as the patients had already established an adequate collateral venous circulation prior to irradiation they suffered little inconvenience. This policy of irradiation followed by exploration would appear at the present time to be the one most likely to lead to success.

Dr. Ian Macdonald said that Dr. Thomson had advanced evidence to support an hypothesis and had made no claim to propounding a theory. His hypothesis was interesting but would require much more evidence from himself and others before it could either be proven or discarded. To reject it because myasthenia gravis was not seen with Hodgkin's seemed to be confusing the issue.

There were great gaps in their knowledge both of the structure and function of the thymus. Dr. Thomson was speaking only of structure and of special cellular structure only. They did not reject a diagnosis of malignancy of the pancreas because they could detect no evidence of disturbance of function. Indeed they should distinguish clearly between structure and function and not attempt to draw conclusions about the effect of one on the other unless their knowledge of their interrelationship was firm. In the case of the thymus there was no such understanding and any discussion of Dr. Thomson's paper should adhere to fact and reasonable hypothesis and not become diverted by improbable analogies.

He could not agree with one statement (in discussion) that Hodgkin's disease does not affect C.N.S. or bone: it does.

Section of Physical Medicine

President—J. SHULMAN, M.B., Ch.B.

[*November 9, 1955*]

DISCUSSION ON THE CLINICAL AND ELECTROMYOGRAPHIC ASPECTS OF POLYMYOSITIS

Professor F. J. Nattrass:

Polymyositis: Clinical Aspects

Among the very varied cases of flaccid paralysis it is necessary to recognize a group in which weakness and muscular wasting are due to diffuse inflammatory changes in muscles. Though a syndrome of polymyositis has been known and described by many authors for nearly a hundred years, renewed interest in it is warranted for two reasons:

(1) The frequent association of such muscular disease with skin changes—dermatomyositis being no longer a very uncommon diagnosis—and the recognition of the affinity of these cases with others of the so-called collagen-vascular or connective tissue diseases, especially scleroderma and lupus erythematosus.

(2) The recognition that polymyositis may occur with no skin changes, or with minimal skin changes, and that the clinical picture then closely resembles that of primary muscular dystrophy. Though such cases are uncommon, they are not so rare as to be unimportant, for the very practical reasons that the prognosis and treatment may be quite different from those of true muscular dystrophy. They present a fairly well-defined clinical picture and a histological picture in a muscle biopsy which is usually distinctive. They have a varied course, sometimes rapid in onset and progress, sometimes insidious and chronic; and they may occur either in childhood or in adult life. They can be separated clearly from septic or parasitic infections of muscles, e.g. trichiniasis, but their ætiology is as obscure as that of collagen-vascular diseases generally.

In trying to define the clinical pictures I shall deal first with a group of cases in childhood which are probably of this nature.

With the co-operation of Dr. John N. Walton I have recently studied, and published an account of, 8 children who recovered from an illness diagnosed in each case as muscular dystrophy (Nattrass, 1954). I shall mention some details of one or two only and summarize the others.

Case I.—This boy was normal till the age of 3 when he began to waddle from side to side and his abdomen stuck out when he walked. He tended to stumble and fall very easily and once on the floor he was unable to get up again by himself. Whereas previously he could climb stairs he now had to pull himself up by holding the banisters with his arms. Within a month of the onset of these symptoms his calves began to swell, particularly the right one which ached a good deal. I saw him at this stage and made the diagnosis of muscular dystrophy. He showed wasting of the pectorals, serrati, infra- and supra-spinati, and looseness of the shoulder girdles. There was marked lordosis with characteristic "climbing up the legs", the thigh muscles were weak and those of the calves were hypertrophied. Within a year the condition had progressed so much that he was unable to walk more than 100 yards by himself and had to be carried to school. The condition remained relatively stationary for another three months and then began to improve.

Constant encouragement on the part of the parents was a feature of his management. At the age of 6 he was able to run races at school. At the age of 11 he lived a perfectly normal life including football and gym, though it was perhaps true that he could not run as quickly as many boys of his own age. Examined in 1952, he was a healthy-looking boy who walked and ran perfectly well and rose from the floor without difficulty: in a word, nothing abnormal was found on detailed examination.

There can be no doubt of the diagnosis of muscular disease. However, it is very uncommon for myopathy to progress so rapidly that the patient is practically unable to walk within a year of the onset, and this relatively acute course is the most important atypical feature.

Case II.—This boy was perfectly well in the first three years of life. At the age of 3 he developed a disinclination to play with boys of his own age because he was unable to keep up with them. He was inclined to play on the floor and when he picked up a cup he used both hands, or put one hand under the other elbow to lift an object. Within three months he was unable to walk more than 50 yards, could not get up from the floor without assistance and was unable to climb a step 6 in. high. He then showed a waddling gait, excessive lumbar lordosis, weakness and wasting of shoulder girdle muscles, and pseudohypertrophy of the hamstring and calf muscles.

This boy was given a vitamin-B preparation for six months and later vitamin-E capsules. Improvement was observed within two to three weeks of beginning the first of these remedies and continued gradually but steadily. It was about eighteen months before he could walk upstairs easily and rise from the floor normally. In time he recovered virtually completely, to lead a normal active life, with only a trace of residual muscular wasting. As in the first case, the rapidity of the onset was, in retrospect, the feature most unlike true muscular dystrophy.

Of the remaining 6 cases, 3 appeared to skilled observers to be the subjects of pseudo-hypertrophic muscular dystrophy. The other 3 were in various respects less typical, though the diagnosis of dystrophy was made in all. In retrospect 2 may have been suffering from a form of benign congenital myopathy. 3 patients began to improve so soon after beginning treatment with vitamin-E products that it seems likely that this treatment influenced the disease. In the other patients recovery was probably spontaneous. In all recovery was virtually complete. There is no proof from muscle biopsy that these 8 patients, whose histories have been traced, and most of whom have been examined by us only since their recovery, were the subjects of polymyositis. Except, however, in 2 of the patients, this diagnosis is the most probable on the analogy of other cases seen subsequently in an acute phase of a similar illness. Certainly they were not the subjects of true muscular dystrophy.

I have seen several cases in adults which have been shown by biopsy to be suffering from polymyositis: in these there is a general similarity in the clinical picture, but with some important differences from the childhood cases.

Patient (Fig. 1) was seen at the age of 50 with a three-year history of increasing loss of power in the arms and shoulders. On examination, there was symmetrical wasting of the scapular and upper arm muscles and bilateral wasting and weakness of the face. There was slight weakness of the spinal and pelvic girdle muscles. He was thought to be a case of facioscapulohumeral dystrophy
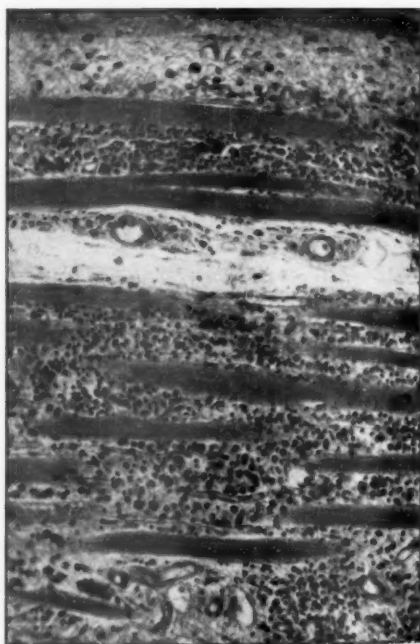
FIG. 1.   FIG. 2.

but with some very unusual features. There was no family history of any similar condition: slight wasting of the sternomastoids and hands was observed but there was little weakness of these. Above all the history was very short and the progress of the condition much more rapid than in this most benign form of muscular dystrophy: deterioration was indeed becoming serious (Fig. 1). An electro-myogram showed changes consistent with a primary muscle disease and a muscle biopsy from the deltoid showed a florid picture of a subacute myositis (Fig. 2). Dr. Walton will deal with the histological changes and the relation to other connective tissue diseases.

This patient improved at once on cortisone. While much of the improvement in his appearance was attributable to the general effects of the hormone, there was also marked improvement in muscular power. This proceeded slowly for several months on a maintenance dose of 75 mg. daily, but at the present time, twelve months later, improvement seems to have stopped and muscular weakness is still severe.

I am left with the impression that adult cases of polymyositis resemble the facioscapulo-humeral type or the limb-girdle (i.e. Erb) type of muscular dystrophy rather than the Duchenne or pseudohypertrophic type of childhood dystrophy. They differ from these forms of primary muscular dystrophy in that they may show involvement also of the muscles of deglutition, of the neck muscles, and to some extent of the hand muscles. A hint is sometimes given of the link with collagen-vascular diseases by a history of vascular pheno-mena of the Raynaud type. A clinical point which I cannot explain is that until an advanced stage, unlike muscular dystrophy, the tendon reflexes may be not only retained but unduly active.

Diagnosis is not rendered easier by the fact that true muscular dystrophy may appear first in adult life, but nevertheless it is likely that more and more cases so diagnosed may prove to be suffering from polymyositis. This conclusion almost certainly applies to the cases described by Shy and McEachern (1951) as "menopausal muscular dystrophy", in which great improvement followed cortisone or wheat-germ oil therapy. It may well apply also to many other cases of apparently typical muscular dystrophy in early life which have been reported as greatly improved after treatment with wheat-germ and/or vitamin E or its analogues. There is very strong evidence that such treatment is entirely without effect in true muscular dystrophy (Walton and Nattrass, 1954).

Conclusions.—Polymyositis without involvement of skin or blood vessels occurring in childhood has, on the whole, a good prognosis and may be influenced by treatment.

Polymyositis in later life has a less favourable prognosis, but seems divisible into two groups:

(1) Resembling a chronic muscular dystrophy and remaining stationary or deteriorating slowly over many years.

(2) Associated with dermatitis and then often a much more severe illness, of shorter duration and high mortality.

Important aid in diagnosis from neuropathic disorders is given by electromyography: Dr. Bauwens will deal with the extent to which this investigation can differentiate poly-myositis and muscular dystrophy.

I would by no means advocate even so relatively harmless a procedure as muscle biopsy in cases of typical muscular dystrophy with a family history. But when there are unusual features, such as a negative family history and especially either an unexpectedly rapid onset and course or the occurrence of remissions, then this aid to diagnosis is essential. In acute or subacute cases the histological picture is usually unmistakable; in the chronic cases the changes are more difficult to identify, but are generally distinctive unless destruction of muscle is very advanced.

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Dr. John N. Walton:

Polymyositis: Diagnosis, Pathology, Prognosis and Treatment

Although progressive muscular dystrophy has been recognized for almost a century as the classical example of a myopathic disorder, we have more recently seen descriptions in the literature of a large number of inflammatory, degenerative and metabolic disorders of muscle, some of which are apparently new. Among these we may mention dermatomyo-sitis, polymyositis, neuromyositis, interstitial myositis in association with collagen disease, generalized myositis fibrosa, calcinosis universalis, menopausal muscular dystrophy and carcinomatous myopathy. Each of these conditions has been described in some detail, but in many instances the number of reported cases is few, and for this reason they are not well known. As Professor Nattrass has pointed out, this lack of general recognition is clearly shown by the fact that cases of polymyositis continue to be diagnosed as progressive muscular dystrophy. Moreover, definition of each of these conditions is often so inexact

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that one cannot determine whether we are dealing with a number of distinct diseases or simply with several variants of a few basic disorders of muscle.

It was with the object of reviewing critically some of these problems that I collected together, in collaboration with Dr. Raymond D. Adams of Boston, a series of 40 cases of what we have chosen to call "polymyositis". Clinically, all of the cases showed, at some stage of the illness, unmistakable symptoms and signs of muscle disease, with weakness and/or atrophy. Pathologically, all 34 cases in which muscle sections were obtained revealed a degenerative and/or inflammatory process unlike that of progressive muscular dystrophy. These observations have led us to conclude that the many conditions enumerated above are variants of a single clinical and pathological syndrome, namely, polymyositis.

We do not use the term polymyositis to imply that the disease is infective, or indeed that it is always inflammatory in nature; in certain cases there is undoubtedly evidence of an inflammatory reaction in the affected muscles, but in others which may be clinically identical such signs are absent. It may seem difficult to justify our terminology in describing cases of the latter type, but the terms dermatomyositis and polymyositis are now so well established that it would be difficult to change them; furthermore, it would not be satisfactory to call the condition "idiopathic polymyopathy", as this description would be equally apt for cases of muscular dystrophy. We feel, therefore, that the syndrome is best referred to as polymyositis while admitting that the term may be a misnomer in certain cases which show no inflammatory change. It is, of course, reasonable to continue to use the title dermatomyositis when there are skin changes or to qualify the diagnosis as, say, polymyositis with rheumatoid arthritis, when there are associated signs of a collagen disease.

Though some workers use the term polymyositis simply to identify a pathological picture, the pathological changes in muscle in the disease under discussion, though generally different from those of muscular dystrophy, are not distinctive, but may be seen in experimental vitamin-E deficiency, virus infections, poisoning with certain toxic agents and even, in very minor degree, following long confinement in bed. It is the combination of the characteristic clinical and pathological features which identify the syndrome of polymyositis, and we believe that the term should be utilized only to indicate this syndrome. The pathological change alone may properly be called a "myositis".

CASES

We have divided our cases arbitrarily into 4 clinical groups which are:

Group I.	Polymyositis.	Childhood. Young adults. Late life ("menopausal muscular dystrophy"). Acute, with myoglobinuria.
Group II.	Polymyositis with muscle weakness the predominant feature but with associated features of a collagen disease	
	or	
	Dermatomyositis with predominant muscular weakness and with minimal or transient skin changes.	
Group III.	Collagen disease with muscular disability of secondary importance	
	or	
	Dermatomyositis with florid skin changes and less obtrusive muscular involvement.	
Group IV.	Polymyositis ("carcinomatous myopathy") or dermatomyositis in association with malignant disease.	

I do not mean to imply that each group is a clearly-defined nosological entity; but the groupings are useful for clinical purposes. During the course of a single illness one may see cases move from one group to another. Indeed, we had one patient who suffered episodes variously diagnosed as acute rheumatism, lupus erythematosus, dermatomyositis, calcinosis universalis and scleroderma, over a period of eight years. The kaleidoscopic course of such cases emphasizes the relationship that most cases of polymyositis bear to the other collagen diseases.

Group I.—There were 14 cases in Group I, of which 9 had been diagnosed as progressive

muscular dystrophy and 2 as myasthenia gravis. 3 patients suffered a very acute illness with severe muscular pain and rapid weakness, developed myoglobinuria and died of renal failure. The pathological changes were those of polymyositis and not of idiopathic paroxysmal myoglobinuria. It seems that any condition in which rapid destruction of large amounts of muscle occurs may produce myoglobinuria, which may, in turn, be fatal because of renal damage. However, 9 of the remaining 11 cases had symptoms characteristic of weakness of proximal muscles in the upper and/or lower limbs—symptoms which were often identical with those of muscular dystrophy. Similar muscular weakness was professed by 2 other patients, but the addition of dysphagia, diplopia, fatigability and an undoubted response to Prostigmin resulted in a diagnosis of myasthenia gravis. Undoubtedly some cases in all groups may show a temporary improvement in muscle power after an injection of this drug. 2 patients were children who climbed up their legs and had pseudohypertrophy of the calves, another was a young adult with a picture like limb-girdle dystrophy (Walton and Nattrass, 1954) and 5 fitted the description given by Shy and McEachern (1951) of "menopausal muscular dystrophy". 2 had facial weakness and 2 involvement of the external ocular muscles. None of these cases showed skin changes, constitutional upset or even significant muscle pain and tenderness; in some the course of the disease was slowly progressive, though in others there were features (such as rapid progression, dysphagia and neck weakness, as mentioned by Professor Nattrass) which are not seen in true dystrophy.

Group II.—Of the 12 cases in this group, 7 were initially diagnosed as cases of muscular dystrophy, generally because skin changes or the associated rheumatoid arthritis were transient or unobtrusive. In the children particularly, the skin changes were often limited to the face and hands, and could be nothing more than a tight, shining appearance of the skin; occasionally there were cutaneous ulcers and minimal subcutaneous calcification. Often the skin changes were those of localized scleroderma or acrosclerosis and the associated dysphagia and Raynaud phenomena had been overlooked.

Group III contained 8 cases, either of florid dermatomyositis or of severe rheumatoid arthritis with secondary polymyositis. In passing, it is worth noting that the skin changes could be very variable, looking like exfoliative dermatitis, seborrhœic dermatitis, lupus erythematosus or scleroderma in different cases.

Group IV.—We have separated the 6 cases in Group IV purely on the basis of an association with malignant disease. 3 were suffering from florid dermatomyositis (like Group III), 1 from severe polymyositis with moderate rheumatoid arthritis (like Group II) and 2 from polymyositis without skin change (like Group I). These last 2 cases correspond closely, from the clinical standpoint, to the condition which Henson, Russell and Wilkinson (1954) and Heathfield and Williams (1954) have called carcinomatous myopathy.

The pathological features are similar in cases of all groups but, as already mentioned, they are not specific, unless combined with the characteristic clinical findings. First one may have variation in fibre size and central migration of sarcolemmal nuclei very like that seen in progressive muscular dystrophy. This occurs particularly in chronic cases or in those which have run a remittent course. Secondly, and most characteristic, there is necrosis and active phagocytosis of muscle fibres, sometimes involving the whole fibre, but more often a segment of it. Thirdly, one usually discovers cellular infiltrates but these may be scanty or even absent (particularly in some Group I or Group IV cases). Finally, there is generally evidence of muscular regeneration; in sections stained with hæmalum and eosin or phloxine-methylene blue the regenerating fibres are basophilic, contain greatly enlarged sarcolemmal nuclei with prominent nucleoli, and show a coarse "granularity" in cross section owing to dispersion of the regenerating myofibrils. This coarsely "spotted" appearance is more clearly seen in sections stained with phosphotungstic acid hæmatoxylin, in which the regenerating fibres are pale.

Prognosis and treatment.—In all groups except the cases with malignant disease, some patients recovered or improved spontaneously, in others the disease became arrested and the patients could lead useful lives, while only a few severe cases died (particularly some in Group III). In all groups, too, some cases responded to ACTH or cortisone. Hence diagnosis from muscular dystrophy is of the greatest importance and often this can only be achieved with certainty by muscle biopsy, though electromyography may help. Our inclusion of all these varied cases under one nosological heading, namely, polymyositis, must not be taken to infer a common ætiology. Although many cases in all groups are clearly related to the other collagen diseases, this is probably not true of all. Our analysis suggests that those cases which show striking cellular infiltrates in the muscle respond best

to cortisone or ACTH; probably in these individuals a hypersensitivity or allergic response is the cause of the disease. In cases without cellular infiltrates, however, which often show no response to similar treatment, it seems possible that the condition may result from some unidentified metabolic or toxic disturbance.

I am indebted to Dr. Raymond D. Adams of the Neurological Service, The Massachusetts General Hospital, Boston, Mass, and to Professor Nattrass for permission to report this material which will be published in detail elsewhere (Walton and Adams, 1956). This work was begun during the tenure of a Nuffield Foundation Fellowship in Neurology; its completion was aided by a grant from the Muscular Dystrophy Association, of America, Inc.

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Dr. P. Bauwens:

Variations of the Motor Unit

I should like to discuss some of the significant electrological vagaries of the motor unit when pathology visits its distal portion. Some cases make history even in the field of electrodiagnosis, and I hope to be forgiven if I outline the one which did so in respect of polymyositis.

Some six years ago, Dr. C. C. Worster-Drought referred to me a typist aged 19, complaining of bilateral weakness of the shoulder-girdles and arms. The condition was said to have started approximately a year earlier with swelling and redness of the palms of the hands, followed by dysphagia and muscle wasting. At the time, the differential diagnosis seemed to lie somewhere between pre-thyrotoxic myopathy and dermatomyositis, with post-scarlatiniform polyneuritis as another suggestion.

Electrodiagnostic investigation revealed that nerve-trunk stimulation in the affected parts produced impaired responses which were well in keeping with the degree of wasting and clinical weakness of the muscles. On direct stimulation of the weak muscles with currents of long duration, sluggish responses were obtained and the intensity-duration curves pointed to partial denervation. As was anticipated from these findings, electromyographic exploration revealed fibrillation at rest, but what was unexpected in the circumstances, and seemed irreconcilable at that time, was the appearance on volition of a motor unit activity pattern which disturbed the whole base line of the cathode ray tube with potentials of low amplitude and short duration—in fact, what might have been expected in a true myopathy, where a large proportion of muscle fibres became inoperative within motor units through primary dysfunction of the muscle fibres.

It seemed here that the impaired activity of a large number of muscle fibres was due to scattered denervation within the motor unit. The phenomenon could be explained by postulating the existence of a pathological process affecting the terminal non-myelinated portions of the ramified neuron causing axon degeneration distally. We labelled this hypothetical condition "distal neuronitis" and looked for other cases presenting similar electrodiagnostic features.

In retrospect, it is now moderately certain that the case just outlined was one of "polymyositis" or perhaps more precisely of "neuromyositis".

Dr. Worster-Drought states that his case proved fatal and that Dr. Peter Daniel of Oxford, on a biopsy specimen, reported that the muscle showed a severe degree of fibrosis with a fairly heavy infiltration of inflammatory cells—mainly lymphocytes but some large mononuclears as well. It appears that the muscle fibres showed a great diversity of size, unusually small fibres predominating and that some of these small fibres had lost their staining reactions and also to some extent their cross striations, while many were broken into short lengths. On the question of intramuscular nerve fibres and endings, the report stated that while a few normal nerve trunks were observed, no nerve endings at the motor end plates were seen.

Since that time, similar cases have been observed, mostly with purely myopathic changes, but sometimes with a neuropathic element so scant that it could not be detected by plotting the intensity-duration curves or testing for nerve conduction and was revealed only on most searching electromyographic exploration by the presence of a few fibrillating muscle fibres.

Frequently, this entity was associated with other pathological processes, as unrelated as carcinoma of the bronchus, steatorrhœa, diabetes and thyrotoxicosis in relation to which it appeared to be secondary. When the primary cause was removed, the electromyographic pattern on volition reverted to an interference pattern compounded of potentials of normal amplitude and duration (Fig. 1).
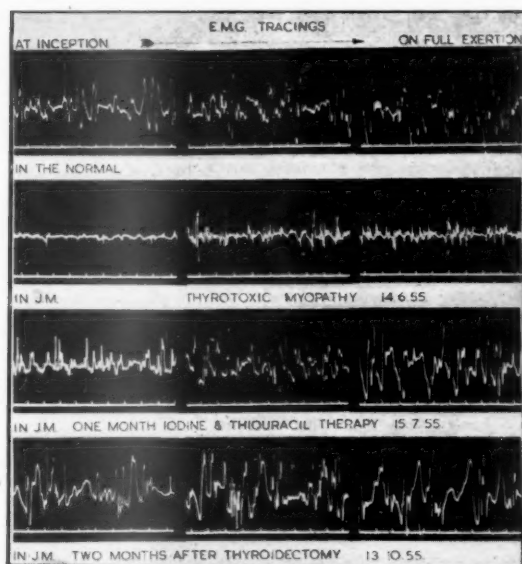
FIG. 1.—Electromyographic tracings obtained with concentric needle electrode from triceps muscle during progressively increased activity (*a*) in the normal; (*b*) in thyrotoxic myopathy showing component potentials of low amplitude and short duration; (*c*) the same after one month's treatment with iodine and thiouracil; (*d*) the same two months after thyroidectomy, showing interference pattern compounded of potentials of normal amplitude and duration. Time scale = 10 milliseconds per division.

In this group the readily reversible character of the muscular disturbance suggests the activity of an endogenous toxin acting in the first place as an inhibiting agent, rather than one causing inflammatory or degenerative processes, although I imagine that a more damaging course, resulting in more chronic dysfunction, can ensue in protracted cases. Such a situation prevails even in myasthenia gravis, another reversible condition which, unless checked appears to gravitate progressively towards a permanent impairment of function—tantamount to a secondary myopathy. In the case of a pure polymyositis, the electromyographic tracing on volition is indistinguishable from that of a myopathy whether primary or secondary. It may depart from it where a distal neuronitis coexists and can be detected. It does not necessarily follow that the neuronitis, when present, is demonstrable. It will be appreciated that where the abolition of the muscle fibre activity precedes the damage to the neuron or neurofibril, it becomes impossible to detect electrodiagnostically the characteristic features of denervation, i.e. sluggish response to direct muscle stimulation with currents of long duration; shift of the intensity-duration curves; or fibrillation on electromyographic exploration. In other words, the myopathic element in polymyositis may mask the neuropathic element—the neuronitis being detectable electrodiagnostically only if the muscle fibres associated with the degenerated axons are still contractile and excitable.

On a previous occasion, I drew attention to the fact that in chronic myelopathies the discrete action-potentials of large amplitude might be replaced by broader polyphasic ones resulting possibly from a temporal dispersion of the activity of the individual muscle fibres. I went further, and said that this type of disintegrated potential might at times be difficult to distinguish from the true myopathic pattern. Perhaps it is therefore not out of place to reiterate here that, in myopathies, the small potentials occur repetitively at frequencies approximating those of the normal motor units, while in myelopathies they occur in recognizable discontinuous repetitive trains at those frequencies.

Dr. A. T. Richardson:

Clinical and Electromyographic Aspects of Polymyositis

Polymyositis may be defined as a reaction of striated muscle of unknown ætiology of which the essential lesion is muscle fibre necrosis, generally with some evidence of regeneration, accompanied by a variable infiltration of inflammatory cells (Figs. 1A and B). Whether,
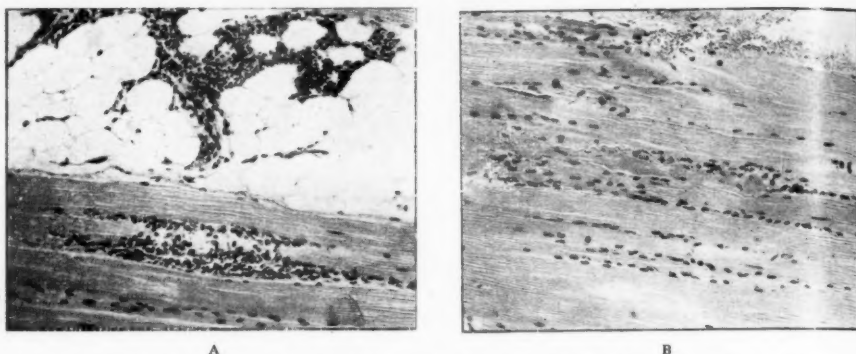
A   B

FIG. 1, A and B.—Muscle biopsy from deltoid muscle showing changes of polymyositis.

in the absence of more exact knowledge of the pathogenesis of this lesion, it can be considered to indicate a primary inflammatory process comparable to that caused by bacterial, viral or parasitic invasion of muscle is doubtful, but there is no doubt that it delineates a muscle disease recognizable not only histologically but often clinically and electrodiagnostically.

In this paper the electromyographic aspects of 20 cases of polymyositis are described with a brief reference to their clinical features. The series is made up of 13 cases referred for routine electromyography and 7 selected classical cases of dermatomyositis which were investigated in addition for abnormalities of neuromuscular transmission.

The 13 cases were referred for electromyography to the Royal Free Hospital and the Hospital for Sick Children, Great Ormond Street, in the last eighteen months. During this period of 486 cases referred, in 52 the presence of a myopathic lesion was reported, this number being made up of the 13 cases of polymyositis, 36 of hereditary muscular dystrophy and one each of thyrotoxic myopathy, periodic paralysis secondary to potassium-losing nephritis, and myasthenia gravis. Further analysis of these 52 myopathies, and I am using the term myopathy in the general sense of a lesion of muscle fibres, shows that in the 21 occurring in adult life the diagnosis of polymyositis was established in 10, while of the 31 occurring in children the incidence was much lower and the diagnosis was established in only 3.

CLINICAL DIAGNOSIS OF POLYMYOSITIS

An early clinical account of polymyositis was given by Steiner (1903) who described it as "an acute, subacute or chronic disease of unknown origin characterized by gradual onset of vague and undefined prodromata followed by œdema, dermatitis and multiple muscle involvement". This description can hardly be bettered except to emphasize that while acute cases frequently involve the skin (dermatomyositis), the mucous membranes or even the peripheral nerves (neuromyositis), in the more chronic forms the myopathy may occur in isolation. Thus apart from the 7 classical cases of dermatomyositis where the diagnosis was made entirely on the condition of the skin, only 5 of the remaining 13 showed skin involvement. However, the co-existence of skin erythema and muscle weakness predominantly in the proximal muscles suggested the diagnosis of polymyositis in 3 cases. Of 2 further cases one had calcinosis of the skin and muscles and the second developed sclerodermatous changes in the skin thus indicating the true nature of the muscle wasting. The remaining 8 cases posed the question of the differential diagnosis of muscle wasting and weakness. In 1 the acute onset of the weakness and wasting with fever, muscle pains and tenderness immediately suggested the diagnosis of polymyositis and in a further 2 the association of proximal muscle weakness and dysphagia did so. In 5 cases the diagnosis of polymyositis on clinical grounds was not made, 3 of these were regarded initially as cases of motor polyneuritis, 2 were regarded as forms of hereditary muscular dystrophy.

The inclusion in this series of the case showing sclerodermatous changes of the skin seems justified because the polymyositis dominated the clinical picture, but I have excluded from this series one case of lupus erythematosus and one of progressive systemic sclerosis in which muscle involvement, although marked, was overshadowed by the other features of these diseases. These cases do, however, illustrate the difficulty occasionally encountered in fixing a diagnostic label to members of the collagen group of diseases showing muscle involvement. It is noteworthy, however, that in all of 3 cases of periarteritis nodosa with weakness and wasting that I have examined electromyographically, a pure lower motor neurone lesion was found and in none could I find evidence of an accompanying myopathy.

Although the presence of Raynaud's phenomenon, raised E.S.R. and increased urinary creatine were an aid to diagnosis, these changes are neither constant nor specific.

The association between polymyositis and underlying malignant disease has been emphasized by Denny-Brown (1953) and others. It is, therefore, of interest that 3 of the 13 cases seen in the last eighteen months have developed a carcinoma of the lung and in one, in spite of repeated investigation, five months elapsed after the diagnosis of polymyositis was made before the carcinoma was detected.

ELECTRODIAGNOSTIC FEATURES OF POLYMYOSITIS

Of the total series of 20 cases of polymyositis examined electromyographically 9 showed a purely myopathic lesion and 8 showed the picture which I believe to indicate a combined lower motor neurone and muscle fibre lesion, i.e. a neuromyopathy. At first examination one case of acute polymyositis showed no abnormality but signs of a myopathy developed two months later and 2 of the 7 selected cases of dermatomyositis were within normal limits.

Criteria of a Myopathic Lesion

The electrodiagnostic criteria of a myopathic lesion are: brisk responses of muscle to direct stimulation, a high rheobase (10 mA or more) and a normal intensity-duration curve as indicated by 100/1 msec. ratios of less than 2. Electromyographically the findings are no spontaneous activity and on volition an increase in the number of short duration and polyphasic motor unit action potentials which on maximal volition build up to a full interference pattern. This electromyographic change is by no means easily detected, the difficulty being the determination of a significant increase of short duration motor unit potentials. With standard equipment it is particularly difficult, but there are two advances now coming into general use which promise to overcome this difficulty. First, by the use of magnetic tape recording the duration of a number of motor unit potentials sampled from a muscle can be accurately measured. Second, by the use of a frequency analyser an estimation of the number of short duration and polyphasic potentials which are indicated by an increase of the high frequency component (over 300 cycles) can be made (Fig. 2).
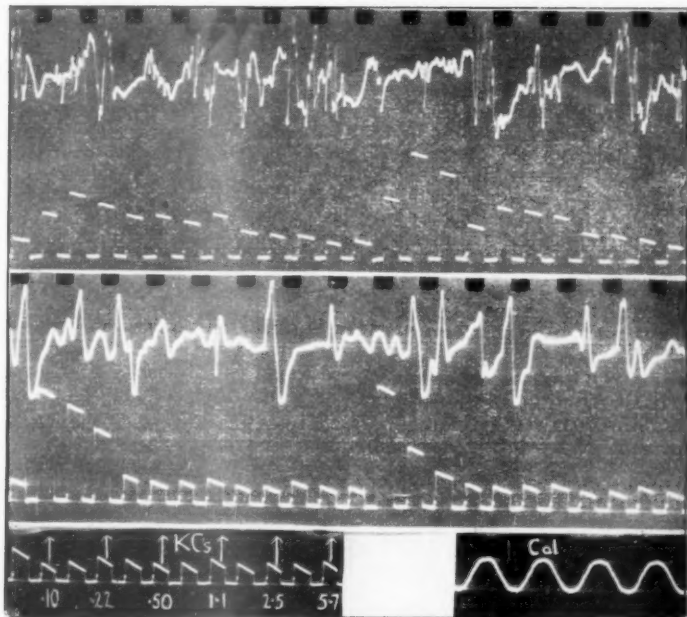

FIG. 2.—Electromyographic tracings and corresponding frequency analysis from: *Top*—Deltoid muscle in a case of polymyositis. *Bottom*—Deltoid muscle in partial lower motor neurone lesion. Calibration is 50 cycles at 100 microvolts. Values of the frequency analyser shown below scale in kilocycles per second.

Criteria of a Neuromyopathic Lesion

The occurrence of cases of muscle wasting in which the electromyographic finding of short duration motor unit potentials characteristic of a myopathy was combined with the classical signs of lower motor neurone degeneration, i.e. fibrillation potentials and/or change of muscle excitability as demonstrated by intensity-duration curves, has been recognized for some time. Bauwens (1949) originally referred to this condition as distal neuronitis and postulated a lesion in the lower motor neurone distal to its point of branching. I think, however, there is little doubt that these cases are in fact cases of polymyositis, for not only did 8 of my 20 cases show these features, but since recognizing the association I have been unable to demonstrate these findings in any other variety of neuromuscular disease. A somewhat similar electromyographic picture is obtained at the stage of early recovery from lower motor neurone degeneration. However, repeat examination at a later date will allow differentiation of the two conditions by virtue of the fact that recovering lower motor neurone lesions soon exhibit long duration polyphasic potentials (10 msec. or longer) which are never seen in a myopathic lesion.

NEUROMUSCULAR TRANSMISSION IN POLYMYOSITIS

The occurrence in cases of polymyositis and dermatomyositis of increasing weakness on exertion suggestive of myasthenia, the apparent response of such muscle weakness to Prostigmin and the demonstration of the pathological changes of myositis in myasthenia gravis (Stortbecker, 1955) prompted an investigation of neuromuscular transmission in polymyositis. Dr. H. C. Churchill-Davidson and I, therefore, measured the response of muscle weakness in 7 selected cases of dermatomyositis, 1 case of lupus erythematosus and 2 cases of acute polymyositis without skin involvement, all of whom complained of excessive fatigue. The method used was measurement of the response of muscles to decamethonium iodide (Churchill-Davidson and Richardson, 1953). In all cases the response was normal, the cases producing the usual depolarizing block and in no instance a block of the competitive inhibition type such as occurs in myasthenia gravis. Similarly we were unable to demonstrate in these cases any convincing response to Prostigmin or Tensilon. It would appear, therefore, that any abnormality of neuromuscular transmission of the myasthenic type in polymyositis, if it occurs at all, is uncommon and these results certainly do not suggest a basis for a diagnostic test.

In summary, therefore, it appears that the diagnosis of polymyositis is readily made on clinical grounds if the muscle wasting is accompanied by the typical skin involvement (dermatomyositis), desquamating erythema, sclerodermatous changes or calcinosis. However, it is the combination of clinical investigations and electromyography that detects this lesion in the majority of cases. Thus the diagnosis can be firmly made if the wasted muscles exhibit the electrodiagnostic criteria of a neuromyopathy and it is strongly suggested if the muscle wasting, shown electrodiagnostically to be a myopathy, occurs in adult life or in the distal muscles or is accompanied by dysphagia or signs of muscle inflammation. There would, however, appear to be a need for more sensitive electromyographic technique to detect myopathic lesions and it is to be hoped that improvements in the method of frequency analysis will supply this.

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Clinical Section

President—Sir HENEAGE OGILVIE, K.B.E., D.M., M.Ch., F.R.C.S.

[*November* 11, 1955]

Cryoglobulinæmia and Lymphoid Leukosis.—A. BATTY SHAW, D.M., M.R.C.P. (for D. N. DOBBIE, F.R.C.P.Ed.).

A. H., male, aged 53. At a routine eye examination in October 1952 he was found to have a bilateral choroiditis; investigations then showed W.B.C. 14,000 (lymphos. 93%) and that 86% of the white cells in his sternal marrow were lymphocytes. He remained in good general health until October 1953, when he first noted swelling of his ankles and nocturnal frequency. In April 1955 the œdema became more extensive, albuminuria was found for the first time and he was admitted to hospital for investigation.

Past illnesses.—1939, iritis. 1942, right-sided thoracoplasty for pulmonary tuberculosis.

On examination.—Pitting œdema of sacrum and ankles. Right thoracoplasty scar. Small areas of bilateral choroiditis, unchanged since 1952. No enlarged glands. Firm enlargement of liver to three fingerbreadths below costal margin. B.P. 180/110.

FIG. 2.
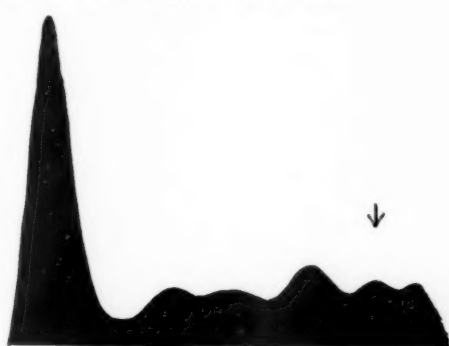
FIG. 1.

FIG. 1.—Electrophoretic pattern of plasma proteins showing high peak in the γ-globulin fraction which is produced by the cryoglobulin.

FIG. 2.—Electrophoretic pattern of urinary proteins showing that this contained the cryoglobulin (marked with an arrow) in the γ-globulin fraction.

Investigations.—Hb 69%. P.C.V. 30%. M.C.H.C. 34%. M.C.V. 88 cu.μ. W.B.C. 18,000 (polys. 30%, eosinos. 1%, monos. 5%, lymphos. 61%, immature lymphos. 3%). Sternal marrow: Slight depression of erythropoiesis, scanty cells of granular series, lymphos. 50·5%, immature lymphos. 3·5%. W.R. negative. Plasma proteins, 8·1 grams/100 ml. (albumin 18·1%, α₁-globulin 3·6%, α₂-globulin 16·8%, β-globulin 7·1%, cryoglobulin and γ-globulin 54·4%, Fig. 1). Liver function tests: Colloidal gold, +4; thymol turbidity 27 units; thymol flocculation, hydrolysed; Kunkel turbidity, 32 units; Popper turbidity, 3 units. Liver biopsy showed numerous deposits of small round cells of the lymphocyte series. Urine: Protein, 3·6 grams/100 ml. (albumin 47·2%, α₁-globulin 10·6%, α₂-globulin 11·5%, β-globulin 14·2%, cryoglobulin and γ-globulin, 16·5%, Fig. 2), hyaline and granular casts, leucocytes and occasional red cells. *B. coli* grown on culture; cultures negative for tubercle bacilli. Blood urea 70 mg., serum cholesterol 230 mg.%. Kidneys concentrate to a specific gravity of 1006 and dilute to 1000. P.S.P. excretion (6 mg. in test dose) 25% excreted in two hours. Urea clearance, 34% in first hour, 24% in second hour. Serum sodium 136, potassium 4·6, chlorides 94 mEq./l., alkali reserve 18 mM./l.

Radiographs.—Chest, right seven rib thoracoplasty. Spine and pelvis, osteoarthritis of lumbar spine. Descending pyelogram, poor excretion by both kidneys.

Comment.—This case is shown for its interest as another example of cryoglobulinæmia, a condition which has aroused much interest in recent years, although there are under twenty-five reported cases in which there has been a high level of this cold precipitable globulin in the serum. Cryoglobulinæmia has most commonly been reported in association with multiple myeloma or cases in which there has been an excess of plasma cells in the marrow (Barr *et al.*, 1950; Nelson and Neill, 1955), but it has also been found in a number of miscellaneous disorders which include kala-azar (Most and Lavietes, 1947) and poly-arteritis nodosa (Lepow *et al.*, 1949). In 1953 Griffiths and Gilchrist reported a case in which it was found in association with lymphoid leukosis (Griffiths and Gilchrist, 1953; Griffiths *et al.*, 1954) and Dr. L. L. Griffiths considered this to be the diagnosis in the present case. Robb-Smith (1938) gives lymphoid leukosis as the synonym for his group of lymphoid medullary reticuloses, to describe the histological change found in the majority of cases showing the blood picture of lymphatic leukæmia. Without entering into a semantic discussion of lymph-node disease, I think there will be those who would prefer to classify the present case as one of lymphatic leukæmia, for it fulfils Willis' (1948) criteria for this diagnosis. Irrespective of the problems of nomenclature, these two cases (only one described here) can be considered to fall into one group, with which can be placed the case of cryoglobulinæmia and chronic lymphatic leukæmia described by Schwartz and Jager (1949).

Patients with cryoglobulinæmia may present with specific clinical features which are secondary to the intravascular deposition of viscous protein in the cold, e.g. Raynaud's phenomenon, purpura on exposure to cold, retinal thromboses, &c. Renal damage may also occur and although this was considered to be a possible cause of the albuminuria in the present case, there was no proof that this was so; this case falls into the group in which cryoglobulin itself is responsible for the production of no clinical symptoms or signs and is found by chance in association with other diseases.

The biochemical aspect is the most interesting aspect of these cases but it is not proposed to discuss this in detail now. Electrophoretic analysis of the serum from the present case, and of the two layers obtained by centrifugation of the cooled serum, showed the cryo-globulin to be present in the γ fraction. The abnormal phenomenon that occurred in the serum thymol turbidity and flocculation reactions would suggest that the cryoglobulin possessed the property of coacervation (Miss V. A. L. Brews); further evidence of this was obtained during the determination of the solubility of the isolated cryoglobulin in saline solution.

(A case of cryoglobulinæmia and lymphosarcoma was also shown.)

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Dr. R. J. Harrison showed coloured photographs of the purpuric lesions on the hands and feet of this patient at St. James' Hospital. This 53-year-old bus driver first noticed small blue spots on his hands and feet on exposure to cold during the winter of 1953–54. The following winter further reddish blue macules appeared on exposure to cold, which formed indolent ulcers. No previous illnesses. Family history non-contributory. Physical examination otherwise negative.

Investigations.—Urine normal. Blood W.R. negative. Blood count normal, no plasma cells. Blood platelets normal.

Test for detection of cryoglobulins.—Blood was collected from the patient, transferred to a test tube, and placed promptly in a water bath at 37° C.; it was allowed to clot; one hour later the serum was separated by centrifuging at 2,000 r.p.m. for ten minutes. On cooling the cryoglobulin spontaneously crystallized from the serum. (A phase contrast photomicrograph ($\times 350$) of the crystals was shown.) Serum proteins: total 7·0 grams% (albumin 4·4, globulin 2·6 grams%). Paper electrophoresis showed a γ-globulin within the mobility range exhibited by normal γ-globulins.

Bone X-rays were normal. Sternal bone-marrow showed 10–15% of plasma cells of normal appearance; there was no vacuolation.

Crystals of cryoglobulin were present in the film, and polymorphonuclear cells were seen with inclusions. (Phase contrast photomicrographs were shown.)

Studies of the cryoglobulins may provide an insight into the underlying metabolic defect which gives rise to their formation. The chief difficulty is isolating them in pure form. The following studies are in progress in this case: (1) The physico-chemical constants of the cryoglobulin. (2) Its

bio-synthesis by the use of isotopic amino acids. (3) Quantitative end group analysis by the fluoro-dinitro benzene method, to determine the number and nature of the terminal residues having free alpha amino group. (4) Its amino acid composition by chromatography after hydrolysis.

It is hoped that such studies will aid in the elucidation of the mechanisms for normal serum protein synthesis.

It was thought that the purpura in this case was mainly due to the cryoglobulin precipitating in the capillaries of those areas exposed to cold. On the other hand, the bleeding tendency is usually a late and uncommon feature in dysproteinæmias, such as multiple myeloma, in which a complex impairment of the hæmostatic mechanism may be present. The most important cause is often thrombocytopenia due to plasmocytic infiltration of the marrow; but renal and hepatic function may also be impaired. The excess globulin may infiltrate the vessel wall, causing increased vascular fragility, and may also interfere with the formation of thromboplastin and fibrin.

Development in a Burnt Child of Antibodies Following Skin Homografts.—PATRICK CLARKSON, M.B.E., F.R.C.S., and PETER GORER, M.R.C.P.

The child was aged 7 two years ago and was admitted eight days after Guy Fawkes night, when she had suffered extensive circumferential burns of her trunk and buttocks involving 25–30% of body surface. She had been treated initially by a full toilet and dressings. On admission she was extremely toxic with a high fever and tachycardia. Dressings were removed, a further toilet was done and cultures taken (which revealed *Pseudomonas pyocyanea*). She was treated by exposure on a Wallace's sectional mattress. Tryptar was used to accelerate the separation of sloughs between the 18th and the 20th days. On the 20th day the remaining sloughs were removed under anæsthesia. Thin split skin grafts cut by electric dermatome from the thighs were placed in strips across her back. These strips alternated with strips of homografts cut from her father (the father was the same blood group as the child). On the 34th day, further autografts were cut by the electric dermatome and applied to the abdomen. All autografts and homografts took well but between the 14th and 20th day after the application of the homografts, they slowly dissolved. On the 48th day after being burnt and the 28th day after the application of the homografts, the raw areas left by their disappearance were covered by split skin autografts. The child was substantially healed about 60 days after being burnt.

About this time Dr. Peter Gorer was able to demonstrate the presence of antibodies in her blood to her father's blood. He stresses the fact that these antibodies may have important late consequences with special reference to the child's future capacity to bear a live fœtus. It is believed that this is the first time that antibodies of this nature have been demonstrated in the blood of burnt patients treated by skin homografts. It may well be that such antibodies only develop in a minority of patients treated with skin homografts but the incidence and the conditions under which such antibodies develop clearly need close investigation. In the meantime and until the incidence of this complication is known, it is very necessary to consider carefully any decision to use homografts in girl children. Homografts may be life saving in the temporary cover they can provide in burns of more than 40% of the body surface with full thickness skin loss. When the area of full thickness loss is less, I believe homografts are best avoided for girl children.

The antibody was first detected by the indirect antiglobulin test. However, rather better results were obtained if the red cells were suspended in compatible human serum and the antibody diluted in 10% dextran (Intradex salt-free). The titre was low but the antibody persisted for at least three months. Similar antibodies found in the sera of mice following homograft regression also persist for relatively long periods.

Both donor and recipient of the graft were group ARh so that it seems unlikely that either the ABO or Rh systems were concerned. The nature of the antigen remains to be determined.

Phæochromocytoma. Malignant Hypertension.—G. S. C. SOWRY, M.D., M.R.C.P.

Male, aged 50.

History.—1943: First recollection of paroxysms of palpitations, i.e. slow pounding of heart with blanching of face; infrequent, lasting two to three minutes. 1945: Hæmaturia. No cause found. Retrograde pyelography showed double pelvis to right kidney. No record of blood pressure. Later seen at hospital for palpitations. No record of blood pressure, but from consultant's letter presumed not abnormal. 1946: Persistence of symptoms. Seen by neurologist. Diagnosed syncopal attacks on evidence of symptoms (blanching and bradycardia) and finding of low blood pressure (120/80). 1947: Paroxysms now more frequent. For short period (probably two to three weeks) associated with severe occipital headaches and some sweating during paroxysms only. 1947–1955: Increasing symptoms; more persistent, less paroxysmal. Blood pressure readings from doctor's records—1951: 170/110; 1952: 150/90; 1953: 170/110. May 1955: Brisk hæmaturia four days. Investigated at hospital. Cystoscopy, intravenous pyelography—no cause found. Double ureter and pelvis on right side.

September 1955: Admitted to Edgware General Hospital. Increasing exertional and nocturnal dyspnœa, blood pressure ranging from 200/130 to 240/170. Marked pulsus alternans. Pulmonary congestion. Left ventricular hypertrophy. Fundi: early papillœdema; extensive soft exudates and a few hæmorrhages. Café-au-lait pigmentation right shoulder. Urine: Albumin +; occasional hyaline and granular casts. Blood urea 31 mg./100 ml. Phentolamine test (5 mg. intravenously): Positive. (Fall of blood pressure from 220/140 to 180/110.) Urinary excretion of adrenaline and noradrenaline 0·6 to 0·7 μg. per ml. on two occasions. (Upper limit normal excretion 0·1 μg. per ml.) (Urinary assays made in Professor Rosenheim's laboratory, University College Hospital Medical School) (Fig. 1).
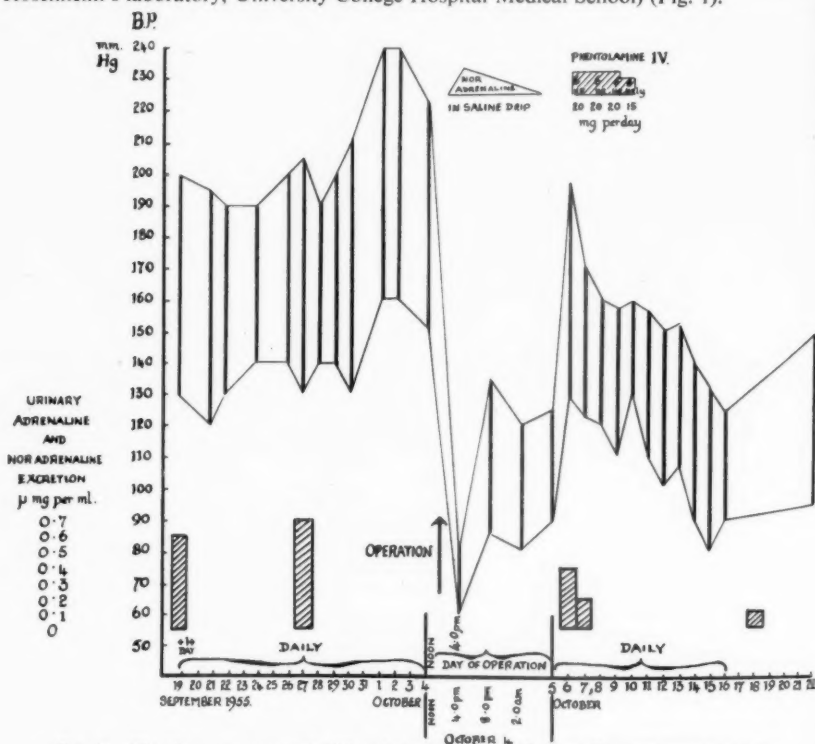
FIG. 1.—Blood pressure record with urinary excretion of adrenaline and noradrenaline.

Treatment.—4.10.55: Mid-line laparotomy (Mr. Noel Slater). Anæsthetic (Dr. J. Attwood): Pentothal, Tubarine, gas and oxygen. Premedication with phentolamine 5 mg. repeated prior to induction. Encapsulated tumour removed from left adrenal, size of golf ball. Right adrenal and abdomen explored. No other tumour found. Biopsy left kidney. Fall of blood pressure controlled with noradrenaline drip for twenty-four hours. Flushing on discontinuing. B.P. 140/90. Gradual rise of pressure and return of cardiac tumult in next twenty-four hours. 6.10.55: Urine assay 0·4 μg. per ml. B.P. 200/135. Six-hourly phentolamine given intravenously, with repeated falls of pressure. 7.10.55: Urine assay 0·2 μg. per ml. 10.10.55: B.P. 160/110. Condition improving. Phentolamine discontinued. 13.10.55: B.P. 140/90. Fundi: no change. 18.10.55: Urine assay 0·1 μg. per ml. 22.10.55: B.P. 150–135/100–90. (Pulsus alternans.) Symptom-free. No dyspnœa. No sweating. Fundi: papillœdema no longer present.

Tumour.—Classical phæochromocytoma histologically. Weight 23 grams. Encapsulated. Renal biopsy: Necrotizing arteriolitis (Figs. 2 and 3).

Comment.—The reason for the post-operative rise of blood pressure with the return of symptoms and signs, strongly suggestive of the presence of a further tumour, is not apparent. The transient high excretion of adrenaline and noradrenaline is also not explained. There was at this time a good urinary output so that the high excretion cannot have been due merely to a urine of unusually high concentration.

Fig. 2.

Fig. 3.

Fig. 2.—Necrotizing arteriolitis of kidney
(Mallory's trichrome).   × 261.

Fig. 3.—Fibrinoid necrosis of glomerulus
(Mallory's trichrome).   × 537.

Necrotizing arteriolitis has previously been described in the kidney in a case of phæo-chromocytoma by Platt and Davson (1950, *Quart J. Med.*, **19**, 33), but then at post-mortem.   No previous record of such a finding at renal biopsy is known to exist.

Malignant Hypertension from Unilateral Tuberculous Kidney Treated by Nephrectomy.—A. E. READ, M.D., M.R.C.P. (for RICHARD ASHER, M.D., F.R.C.P.).

C. G., male aged 17, was admitted to the Central Middlesex Hospital on 4.5.55 with a history of 1 year's frequency of urination, hæmaturia and right renal colic.   There was also deterioration of the sight in his right eye for 3 months due to tuberculoma of the retina.

Fig. 1.—Drawing of right retina to show papillœ-dema and macular exudates and old tuberculoma.
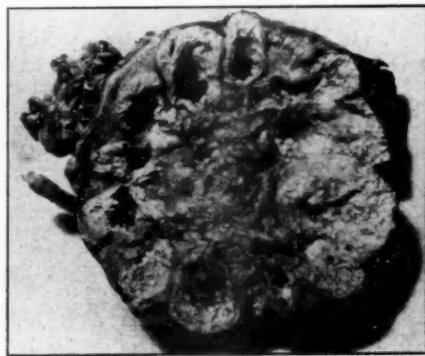

Fig. 2.—Right kidney showing extensive tuberculous involvement.

Past history.—Tuberculous spine when aged 4 years, treated by immobilization and bone grafting.

On examination.—An ill boy, dwarfed by angular kyphosis in mid-dorsal spine. Cardiovascular system: Heart size and sounds normal. B.P. 210/180. Fundi: Right eye at 12 o'clock—patch of choroidoretinitis probably an old tuberculoma (Fig. 1). No exudates or papilloedema seen at this time.

Investigations.—*Chest film:* Large calcified gland in the left upper zone. Blood urea 36 mg.%. Blood count: Mild hypochromic anæmia and a polymorph leucocytosis. Total W.B.C. 13,500. Urine: Two of three specimens grew tubercle bacilli. I.V.P. showed a non-functioning kidney on the right side, the left appearing to be normal. Cystoscopy (11.5.55) showed diffuse tuberculous infiltration of the bladder with inflammatory changes round both ureteric orifices preventing the passage of ureteric catheters.

Soon after cystoscopy he complained of severe headache and vomiting. His blood urea rose to 59 mg.% and frank papilloedema with macular exudates developed (Fig. 1).

It was clear that his hypertension was entering a malignant phase and we believed right nephrectomy was urgently indicated. This was carried out on May 28 after an initial course of Ansolysen by mouth. An extensively diseased kidney was removed (Fig. 2).

Since the operation the blood pressure has stayed within normal limits for six months (Fig. 3); his papilloedema has resolved and apart from developing a cold abscess of the
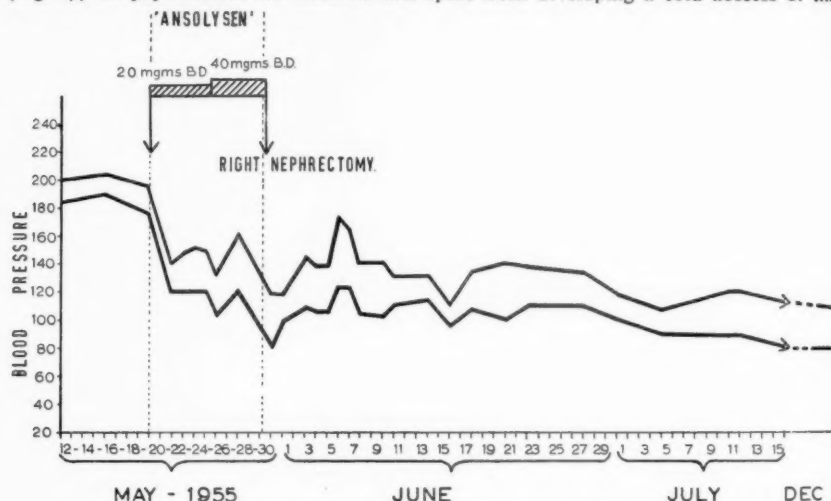
FIG. 3.—Chart of blood pressure from May to December 1955.

sternum he has remained well. Throughout this time he has received anti-tuberculous chemotherapy.

This case is interesting because of the wide dissemination of the tuberculosis (of which evidence could be found in the spine, retina, chest, kidney and sternum), and the dramatic response of the hypertension to nephrectomy. Tuberculosis is not a common cause of renal hypertension. Pickering and Heptinstall (1953) report one case in a series of 11 nephrectomies, the blood pressure of which was not lowered by surgery. Langley and Platt (1947) reported 3 successful cases from the literature in a collection of 93 nephrectomies. Van de Goidsendoven and Vandenbrouck (1946) record 6 cases of renal tuberculosis in which there was hypertension—2 of these responded in some degree to nephrectomy.

Other reviews and isolated cases stress the good response to nephrectomy in patients with renal tuberculosis complicated by hypertension. This is probably because they are usually younger than people with other forms of unilateral renal hypertension.

The excellent response of our patient to nephrectomy despite his malignant hypertension supports such a view.

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BOOK REVIEWS

Genital Prolapse and Allied Conditions. By Percy Malpas, Ch.M., F.R.C.S., F.R.C.O.G. (Pp. 199; 37 figs. 47s. 6d.) London: Harvey & Blythe Ltd. 1955.

In his book on "Genital Prolapse and Allied Conditions", Mr. Malpas has, out of his long experience, given us a work of real value. Here is perhaps no textbook for the undergraduate student reading for an examination in gynæcology, but for the practising gynæcological surgeon, its sound judgment, accurate detail, and, above all, its unbiased sifting of long and extensive operative treatment, will prove of inestimable value.

The scope of the book is wide and an attempt is made to trace the ætiology and development—if one may use such an expression—of the various methods of treatment of genital prolapse and, with this, to evaluate our present position with regard to these.

As is to be expected, the book commences with a chapter on the anatomy of the region involved and an earnest attempt is made to clarify this difficult subject. The gynæcologist, however, who has been operating for many years will find it difficult to change his preconceived ideas on the anatomy of the operation area and his familiar reliance on Mackenrodt's and the uterosacral ligaments, nor do I think that his operative results will necessarily suffer from not doing so.

The chapter, however, dealing with disturbances of micturition associated with genital prolapse is one of the most valuable in the book, for the discussion of the treatment of such symptoms, especially stress incontinence, is preceded by a lucid description of the physiology of micturition on the knowledge of which the rationale of treatment is based. Thus, great assistance is given in the choice of operation in an individual case and the types of operation are clearly described, although here, as throughout the book, illustrations and diagrams are somewhat lacking. Just as valuable, in my opinion, is the discussion of reasons for operative failure to relieve the patient's symptoms and suggestions as to further treatment.

The part of the book dealing with operative treatment is preceded by a most valuable chapter on the ætiology and classification of the varieties of prolapse, this greatly assisting in the planning of the operative measures to give relief. Thus, the ætiology of uterovaginal prolapse of the first or second degree is different from what Mr. Malpas describes as general prolapse, and, while the author's predilection would appear to be for the Manchester-Fothergill type of operation, he maintains an unbiased view and considers that many cases of this second type can only be relieved by a modified vaginal hysterectomy. Some of the more difficult varieties of prolapse to treat, which often receive little attention in the general gynæcological textbooks, such as nulliparous prolapse, prolapse after hysterectomy, are fully considered and the book concludes with a chapter of rectal prolapse and retroversion.

Mr. Malpas is greatly to be congratulated on this volume, which is thoughtfully and lucidly written, and well produced. Embodying as it does not only the results of the author's long experience, but also much study of past and present work on this subject, it should prove of great value to all gynæcologists.

The Extra Pharmacopœia (Martindale). Vol. 2. 23rd edition. (Pp. xxxii + 1501. 57s. 6d.) London: The Pharmaceutical Press. 1955.

The second volume of the 23rd edition has appeared within three years of the first volume; but it is twelve years since the last second volume left the press. The general format is still the familiar one, but the size has once more increased. In its seventy odd years of existence Martindale has expanded from one volume to two volumes, and now contains about nine times the original total amount of material. This volume has been completely revised and several new sections added reflecting the trends of modern interest. The sections on clinical biochemistry and hæmatology, and their interpretation have been greatly expanded. There is now a section on radioactive isotopes, and the section on chromatography is larger. The section on chemical nomenclature still performs its useful function of guiding the novice through this complicated web. It is closely followed by a short review of structure-action relationships which will serve as a useful introduction into this complicated and much debated field. These are only a few of the valuable features. To review it in detail is not the task of a single reviewer, he can only make a few points which interest him more especially. A number of the so-called sections are long enough to form almost small textbooks. Such are the bacteriological and clinical notes, and the sections on hæmatology, and clinical biochemistry, and the recognition of organic chemicals. The editors are to be congratulated on their ability to pack such a colossal amount of useful information into the compass of

this volume and still keep it readable, and abreast of the times. The volume is well up to its old standards. Those who normally have used it in the past will welcome the completion of this 23rd edition. It still remains the best single reference book on the fundamentals of pharmacy and the subjects allied to it, and as such is still to be recommended.

Saddle Block Anæsthesia. By Ray T. Parmley, M.D. (Pp. x + 59; illustrated. 18s.) Oxford: Blackwell Scientific Publications. Springfield, Ill.: Charles C. Thomas. 1955.

The term "saddle block" is applied to that form of low spinal anæsthesia which is restricted to the sacral nerves and thus produces a saddle-shaped area of anæstheisa around the perineum. It is a well established, simple and reliable technique.

In 1946 Dr. Parmley, with Dr. John Adriani, described the use of this method in obstetrics, and the present monograph is based largely on this work together with a description of certain elaborations of the technique which had previously been published by Dr. Adriani and his associates.

The author gives detailed instructions for producing saddle, "modified saddle", and low spinal blocks, and describes their indications and the precautions to be taken. The treatment of any associated hypotension or respiratory failure is considered and Dr. Parmley reviews the complications of spinal anæsthesia but fails to add to our knowledge on the controversial subject of the neurological sequelæ.

Indeed, there is little that is new in this slim volume which is, unfortunately, not free from misprints and contradictory statements—for instance, the legends do not accurately describe Figs. 4 and 5. Like its predecessors in the American Lecture Series, this monograph is excellently produced and bound, and Dr. Adriani contributes a thoughtful and balanced introduction.

Virus and Rickettsial Diseases. By S. P. Bedson, M.D., D.Sc., F.R.C.P., F.R.S., A. W. Downie, D.Sc., M.D., F. O. MacCallum, B.Sc., M.D., and C. H. Stuart-Harris, M.D., F.R.C.P. 2nd edition. (Pp. viii + 407; 34 figs. 30s.) London: Edward Arnold (Publishers) Ltd. 1955.

In preparing the second edition of this text, the authors have firmly resisted the temptation to achieve modernization by expansion. Although new material has been added on Murray Valley encephalitis, the Coxsackie viruses, virus multiplication and cat-scratch fever, an increase in size has been avoided by cutting some of the references to the animal pox diseases, bacteriophage, and other matters of more specialized interest. The volume and importance of recent research on chemotherapy and poliomyelitis have forced a major revision of the discussion of these subjects but a discriminating selection of references to the literature has produced a balanced presentation of current opinion. Throughout, although the authors share a certain compactness of expression, clarity has not been sacrificed to brevity; all that the epidemiologist or clinician might reasonably want to know is there. On the other hand, this short text does not pretend to be an encyclopædic source for the specialist in bacteriology. Within these self-imposed limits, it is unquestionably destined to become a "must" in the library of all who need a reliable guide through a field of ever-increasing importance.

Psychological Medicine: A Short Introduction to Psychiatry. By Desmond Curran, M.B., F.R.C.P., D.P.M., and Maurice Partridge, M.A., D.M., D.P.M. 4th edition. (Pp. viii + 407; 20 figs. 21s.) Edinburgh and London: E. & S. Livingstone Ltd. 1955.

This admirable English textbook has been expanded and large portions of it have been rewritten. It is no longer short, as its title would suggest, but every student will agree that a book must contain a reasonable amount of detail if it is to be useful to him. The book takes what is now becoming the accepted English standpoint, in that it distinguishes psychiatric illnesses which are entities in the Kraepelinian sense, and those which are reaction types or progressive failures of adaptation in the sense described by Adolf Meyer. The statement of this position in the second chapter, which deals with ætiology, is one of the most excellent sections of the book.

The descriptions of various syndromes follow ordinary lines save for the clarity of description and the immaculacy of the English employed. The chapter on the legal aspect of mental illness, which is of great importance to the medical student from the examination point of view, is notable for the detail given in what is a relatively short chapter. Incidentally, the facts given appear to be accurate. There is very little that can be criticized in the book, and the reviewer was only able to find two small points in a fairly close examination of it. The dosages of Scoline on page 358 have been transposed in that a smaller dose is given to a man than to a woman. The other point is that the statement that anxiety states are rare would probably not be true if the Geneva list were to be fairly followed. According to this list all states with tension as a prominent symptom should be included under anxiety states,

whether hysterical features are present or not. These, however, are small criticisms in a book which can be cordially recommended to undergraduates and doctors in any branch of medicine.

The book is attractively produced and has a comprehensive index.

Thallium Poisoning. By J. J. G. Prick, W. G. Sillevis Smitt and L. Muller. (Pp. viii+155; 21 figs. 19s.) London: Cleaver-Hume Press Ltd. Amsterdam, etc.: Elsevier Press, Inc. 1955.

This monograph is the first of its kind to deal in detail with a single poison and it is to be hoped that it is the predecessor of others. Most poisons are dealt with in textbooks of forensic medicine and toxicology, but in limited detail, for space dictates how much can be included and policy that none shall be left out. It is, in fact, necessary to go back to the great Witthaus who wrote "Manual of Toxicology" in 1911 to find a really detailed description of even a common poison such as arsenic.

Thallium is little known in this country except as a therapeutic depilatory agent but it has been used for more harmful objects elsewhere and particularly in Australia. This may be because it is in more common use as a pesticide. This monograph does not limit itself to symptomatology, identification and treatment but includes both pathological anatomy and experimental pathology. The authors modestly excuse themselves for their work on grounds of public interest, but the production has more than justified itself as an academic contribution to clinical toxicology which draws attention to the close resemblance between clinical diseases such as Landry's paralysis and polyneuritis and thallium poisoning, thereby sounding a warning that exclusive chemical analysis may from time to time be advisable.

There is an excellent review of the literature in this book.

Obstetrics. By J. P. Greenhill, M.D. 11th edition. (Pp. viii+1088; 910 figs., 144 in colour. 98s.) Philadelphia and London: W. B. Saunders Co. 1955.

De Lee's famous textbook of Obstetrics appears in its eleventh edition compiled by Greenhill. In its present form it continues to maintain and to advance the deserved reputation of its predecessors. The thousand double-columned pages of which it consists are a remarkable storehouse of sound information, extensively culled, systematically assembled, judiciously considered and admirably presented. The style is lucid, contributing to facile perusal. The efforts of the publishers are also to be commended for the excellent quality of the production.

The main division of the book is into a consideration of the normal and of the abnormal processes; in each the scientific approach to the problems involved is characterized by the wealth and accuracy of the data provided, whilst their correlation to the practice of the art is subsequently well effected.

A noteworthy feature of the book is the collaboration of the author with authorities in several specialized fields, amongst which may be mentioned anæsthesia, erythroblastosis, puerperal infections and disordered renal states. Endocrine physiology and pathology in pregnancy is still another, from which many interesting points emerge: the improvement of hypopituitarism in pregnancy—indicating the manufacture of the missing hormones by the placenta; the augmenting effect of chorionic gonadotrophin on the level of antidiuretic substance in the serum; the reappearance of adrenal steroids in pregnancy in patients suffering from Addison's disease; the failure of ACTH administration in evoking toxæmia in the normally pregnant; the different level of production of supracorticoids in normal and toxæmic pregnancy, and so on. These serve as examples to illustrate the detailed care in producing the section.

The normal processes of labour are also thoroughly dealt with and profusely and aptly illustrated. The diagrams of the pelvic floor need special commendation.

The chapter on anæsthesia, also written in collaboration with an authority, contains an evaluation of oral and hypodermic methods in obstetrics; of inhalation and intravenous agents; and of block and local infiltration techniques. It covers recent advances in the field and gives much attention to hypnosis and to the Grantly Dick Read approach. However, there is no reference to the use of gas and air anæsthesia, an oversight of significance to the British reader.

The three stages of hyperemesis gravidarum are well discussed, but the insidiousness of approach of the third and fatal stage has not been sufficiently stressed.

The section on ante-partum hæmorrhage contains much recent information. Prominence is accorded to Schneider's theory of hypofibrinogenæmia in the ætiology of accidental hæmorrhage. The ready occurrence of shock in these patients is ascribed to the frequently associated and preceding toxæmia, perhaps a controversial point. Although renal artery spasm is included as an accompaniment of the state the threat of subsequent anuria remains

unmentioned. A valuable observation is recorded, that fœtal death is often wrongly presumed to have occurred in cases of severe abruptio placentæ—an encouragement to resort more frequently to Cæsarean section in such circumstances.

The subject of toxæmia is amply discussed in collaboration with an authority on renal dysfunction emphasizing the close ætiological association of the two. The intrusion of the kidney both into this syndrome and into that of concealed accidental hæmorrhage would be further appreciated if the elementary biochemistry of the organ and its hæmodynamic interpretation were more completely explained in subsequent editions. The advance in obstetric education demands this expansion if a proper appreciation of the subject is to be had by all. It is satisfying to note that the modern theses of pre-eclamptic ætiology, such as the myometrial resistance to stretch, are included in the text.

A pretty experiment is cited to exclude uterine pressure on the ureter as a cause of pregnancy pyelitis. It demonstrates that the pregnant monkey can develop a hydro-ureter with the placenta alone in situ after the fœtus has been removed, an indirect proof of a hormonal ætiology and a denial of a pressure effect. Routine microscopy of the urine at every antenatal visit is urged so that the earliest possible diagnosis of pyuria can be effected. These exemplify the meticulous approach into theory and practice once again.

Numerous data of value emerge in the chapter on erythroblastosis, amongst them that kernicterus is not present in the stillborn and that erythroblasts in the macerated fœtus occur solely in lung capillaries. For completeness, the rarer forms of its treatment are discussed. They include immunization with a stronger antibody-stimulating antigen, ethylene disulphonate, methionine, Rh hapten, ACTH and cortisone.

The author of a textbook on obstetrics is always faced with the problem of the presentation without repetition of subjects that are closely interdependent. A single theme may comprise the failure of rotation of the posterior occiput borderline contractions of the pelvis and of its unusual contour or uterine inertia. The failure of the test of labour can succinctly be described as occurring when "progress is seemingly blocked" [*sic*]. Detailed consideration may have previously been given to the processes from which such a diagnosis stems. The spreadover of such information is greater in proportion to the size of the book that contains it, and its clinical educational impact correspondingly suffers. A worth-while solution for the comprehensive presentation of these themes is not herein evident, though the relative information is complete.

Improvement could be effected in certain directions. As an instance, there could be an amplification of X-ray aids in diagnosis; the Liverpool technique of delivery of the after-coming head could be described; and attention could be drawn to the risk to the fœtus when the low-lying placenta is posteriorly sited. Then again, although great thoroughness is displayed in the treatment of post-partum hæmorrhage, criticism may be levelled at the advice to invade the uterine cavity, four times even on occasion, without previous investigation of the blood-clotting mechanism for a possible defect. Also more in keeping with current practice would be a description of the transverse myometrial incision rather than the vertical the author employs in lower segment operations.

An occasional contradiction appears but there are very few typographical errors.

Asynclitism is clearly portrayed in the excellent illustrations of contracted pelves and its implications fully discussed. The diagrammatic and the serial visualization of transverse arrest is extremely well done. Modern advance is evident in that lower segment section is advocated should forceps fail to deliver at trial labour and amongst others that "most primiparæ" should have pelvic roentgenograms shortly before term because of the risk of mid-plane contraction remaining undetected.

Deserving of mention also is the pertinent bibliography; the excellent tabulations, as for example, the drug substances excreted in the milk; the differential diagnosis between pyelitis and appendicitis, and so forth; the proximity of the diagrams to the text they illustrate; the employment of modern methods of treatment such as surgical procedures for mitral stenosis in pregnancy, and so forth. Attention to prophylaxis is also very evident. The exhibition of vitamin K shortly before birth in *all* difficult labours; the employment of oxygen inhalation during parturition to sustain the fœtus; the interdiction of external version should the placenta be anteriorly sited—an implication that X-ray investigation is imperative as a preliminary to such procedures, are among the innumerable injunctions presented.

The book, though expensive, is good value, and can be thoroughly recommended for it is a vast storehouse of sound knowledge accumulated over the years and well presented. Though American in origin it yet admirably serves the British reader. Specialist and post-graduate alike must derive considerable benefit from its perusal. The undergraduate reader will also profit, for the subject matter is simply and clearly presented and well within his competence to appreciate.

Dr. Greenhill, whose reputation for advancing obstetric and gynæcological education is world wide, deserves commendation in producing this, the eleventh, edition.

Section of Proctology

President—HAROLD DODD, Ch.M., F.R.C.S.

[*November* 17, 1955]

The Charter of Proctology

PRESIDENT'S ADDRESS

By HAROLD DODD, Ch.M., F.R.C.S.

MY purpose is to review the contributions of past Presidents to the Section of Proctology, and from them to submit an addition to the Charter of Proctology which was articulated by Professor Grey Turner when he was President in 1925. This means compressing forty-two years into a short paper.

The period since 1913 resolves itself into four parts: (*a*) the First World War; (*b*) the inter-war years; (*c*) the Second World War; (*d*) and the last decade.

Each is characterized by developments in surgery in general and proctology in particular. I propose to sketch the latter.

Proctology in 1913.—Mr. Swinford Edwards was our foundation President from 1913 until 1919. There was healthy rivalry between St. Mark's and the Gordon Hospitals. Two books documented the specialty, that on the Rectum and Anus, by Goodsall and Miles, and Lockhart-Mummery's Jacksonian prize essay on "Diseases of the Colon". Colitis was then largely mucous or membranous, and diverticulitis appeared as pericolitis.

The first published Presidential Address was in 1920, by the surgically immortal Ernest Miles, who spoke on "How soon should a colostomy be performed in cases of carcinoma of the rectum which are inoperable". This subject would scarcely arise nowadays, because the operability rate has increased from 35% in 1913, to over 90% to-day.

A colostomy continues to be an essential life-saving operation. It was discussed in 1950, when a moot point was, whether the patients should wash out their colostomies, or leave them alone; the latter view seems to be winning the day, I have changed to it for patients also prefer it. A colostomy receding into the abdomen still has to be guarded against. I have lost a patient through this occurrence. The suture of the bowel mucosa to the skin, as described by Patey and again by Clive Butler in his 1951 Presidential Address, confirms its usefulness as a good procedure; I do it subsequently at a trimming operation.

The history of proctology.—Three Presidents have described proctology from the earliest times. In 1932, Perrin of the London Hospital spoke of the historical aspects of piles and fistulæ. In 1947 Hedley Whyte, the second President from Newcastle, gave a lively review of proctology past and present, whilst Miller of Edinburgh in 1953 painted a masterly picture of the times of Edward III, the Black Prince, and John of Arderne, who is regarded as the Father of Proctology. He was a general surgeon with unusual skill in abscess, fistulæ, piles, prolapse, condylomata, proctitis, tenesmus, dysentery, polyps and cancer. These ailments have a familiar sound and lead to others mentioned by Grey Turner in 1925, such as congenital rectal and anal defects, syphilis, amœboma, stricture, multiple polyposus, colitis, diverticulitis, non-specific granuloma, constipation and rectal neuroses; the last is notable with its medical and psychological problems. Thus men and their bowel ailments are much the same now as 30 and 600 years ago, but knowledge, remedies and surgeons, we may reasonably say, have grown.

The anatomy of the rectum and colon.—The anatomy has been expounded to us by two distinguished Presidents, Milligan and Morgan. In 1942, Milligan detailed that of the peri-anal region. His work is still fresh and I derived much instruction from re-reading it. Milligan and Morgan's anatomical study published in 1937 was extended by Morgan when President in 1948, with special reference to the pelvic floor and the problems of extensive ischio-rectal abscesses, deep fistulæ and perineal excision of the rectum. Morgan repeated his anatomical exposition to the American Proctologists in 1949 and I well recall Mr. Gabriel urging the younger surgeons there "to master it"; it is an evergreen necessity.

Physiology.—The physiology of the rectum and colon received fascinating and instructive consideration from Sir Heneage Ogilvie in 1950. He said, "Normal defæcation is essential to health and happiness". He emphasized that if a restorative anastomosis of the rectum and colon is to be a success, the reciprocal function of the rectum and anus must be preserved, therefore some rectum must be retained. If all the rectum is excised, control is imperfect,

as in the unconscious incontinence which often follows rectosigmoidectomy for prolapse, as reported here by Hughes and Thompson in 1949 (*Proceedings*, **42**, 1007, 1011). Such a bowel may be a greater liability than a colostomy. The preservation of at least 1½ to 3 in. of rectum, advised by Hughes, Thompson and Muir, is sound practice.

Intestinal diseases.—Aslett Baldwin in his Presidential Address of 1923, elaborated some of the causes of intestinal diseases, based on the premise of unsuitable diet, over-prepared food and the form of modern civilization.

This subject leads to the consideration of the maladies of the anus, rectum and bowel.

Hæmorrhoids.—Why do people develop hæmorrhoids, which, like varicose veins of the legs, occur at all ages and tend to run in families. We know the factors said to predispose to piles by increasing the intra-abdominal pressure, but we all know people who have these conditions and yet do not have hæmorrhoids. Professor Milnes-Walker and Alan Hunt tell me that in their experience of portal hypertension, no case of it has presented with rectal bleeding as the chief symptom. Clearly, high pressure in the portal system, i.e. pressure from above, does not ordinarily dilate the hæmorrhoidal veins. Conversely, a spastic sphincter is not contributory, else one would expect exceptional piles in fissure-in-ano, i.e. obstruction to venous flow is not essential. Might hæmorrhoids be due to small arterio-venous fistulæ? The recent work of Piulachs and Vidal-Barraquer (1953) suggests these latter are causative in some cases of varicose veins. Here is a subject for study, but meanwhile I like the basic explanation that patients with piles are born with too many veins in their anal canal and that various strain and stress factors aggravate this abnormality. Such a thesis explains their occurrence in those in whom no cause is apparent. We are all familiar with the associated varicose conditions such as varicose veins of the legs.

In 1913, the treatment of piles was usually that of excision by Salmon's operation, but this procedure was not generally well done, for Ernest Miles related that when he went to France in the First War, he found 2,000 soldiers sick from piles. He was told that operation was useless because no man so treated had ever returned to duty. Miles undertook to cure patients in a month, and he operated on 15 cases daily until his hutted hospital was bombed.

In the last war, piles were reasonably well done, although Brigadiers Harold Edwards and Naunton Morgan (1955, personal communications) tell me that they saw tiresome complications frequently enough to emphasize the care that must be given to this operation.

Description of technique.—The Milligan-Morgan technique (Milligan *et al.*, 1937) for hæmorrhoids based on the Salmon operation is now classical and certain. It should be acquired and perfected by all. In this procedure the three pile masses are completely excised, beginning with the skin beneath the external part of the pile lying over the lower end of the internal sphincter. This allows the internal and external pile to be freed and pulled down until the neck or pedicle is at the lower end of the anal canal. It is secured here by a strangulating ligature. The piles are not stripped up or a stricture may form. Care is taken to preserve the bridges of mucosa and skin between each pile; this also prevents stricture of the anal canal.

The injection of hæmorrhoids.—Like the injection of varicose veins, the injection method of treating hæmorrhoids came in with this century. It was brought to Britain from America by that scholarly President, Swinford Edwards, who was the last consultant to be on the staff of both St. Mark's and St. Peter's Hospitals. Injections for varicose veins and piles were hailed as "the" treatment, and time has shown their value. Injections of varicose veins now play but little curative part, but submucous injections for piles of the 1st and 2nd degree are the proved and accepted remedy.

The complications of hæmorrhoids.—In 1930 Cecil Rowntree of the Royal Cancer Hospital chose this subject for his Address. He mentioned the sudden loss of sphincter tone which indicated collapse or death: the shock-producing procedure of stretching the sphincter was then routine practice. Severe post-operative pain obtained then and may still do so; we have yet to learn how to control it in all cases. I find that getting patients up within twenty-four hours of the operation for bed-making, washing and toilet purposes is helpful. Pethedine 50 to 100 mg. by mouth four-to-eight-hourly is usually adequate. A fluid diet is given and on the fourth or fifth day a small enema of olive oil and turpentine generally gives a smooth, not unduly painful bowel action. A nurse or house surgeon passing a finger quickly into the anal canal often causes a spate of tears, pain and fears because they do not know the direction of the anal canal or that the sphincter yields almost painlessly to gentle pressure.

Reactionary hæmorrhage after hæmorrhoidectomy is still a possibility—its likely source being the external raw surface remaining after the skin cuts. These are superficial, so that with a good light this bleeding can be secured without an anæsthetic. Bleeding from the pedicle nowadays is rare, although secondary hæmorrhage may occur up to the fourteenth day.

Rowntree also mentioned retention of urine and stricture of the anal canal. By catheterizing my patients before they leave the operation table, post-operative bladder difficulties have largely disappeared.

A constriction of the anal canal varying from slight to considerable may follow the operation and I think that it is essential for every patient to have a finger passed into the anal canal once a week until it enters easily and the rectum is free of narrowing or pain.

Prolapse of the rectum.—Last session Muir (1955) gave us a historical survey of prolapse of the rectum. Two significant points merit recall, first that in spite of the high recurrence rate and incontinence that may follow the Miles recto-sigmoidectomy for prolapse, it is still useful in feeble and elderly patients because it carries little morbidity and mortality and can, if necessary, be repeated. Muir described an improved procedure, an intra-abdominal excision of the redundant rectum and sigmoid, with an end-to-end anastomosis. The shortened bowel adheres firmly to the sacrum and no longer prolapses. It was the fixation of the rectum observed after an anterior resection for cancer that suggested this operation. Both patients and surgeons welcome this radical cure for prolapse as it promises the consistent success which attends other proctological measures.

Carcinoma of the rectum.—The results of treatment of cancer of the rectum are notable. All the Presidents of this Section, especially Miles, Lockhart-Mummery, Grey Turner, Gordon-Watson, Norbury, Dukes, Milligan, Gabriel, Abel, Morgan, Lloyd-Davies and Butler have made distinctive contributions. Dukes' pathological work has copiously illuminated this Section's *Proceedings* since 1920 and it culminated in his analysis of over 1,000 cases of rectal cancer in 1944. The Dukes' classification of rectal carcinoma is now the adopted standard throughout the surgical world. He has established the prognosis with rare accuracy. The follow-up department at St. Mark's Hospital, extending as it does over 34 years, includes 3,500 cancer patients. In 1940, Mr. Gabriel, when President, comprehensively described squamous carcinoma of the anus and its variations based on 55 cases, compared with 1,600 patients admitted in the same period with carcinoma of the rectum (Gabriel, 1941).

The diagnosis.—The responsibility of diagnosis of carcinoma of the rectum is ever with us. It requires investigation of the rectum by the finger, proctoscope, sigmoidoscope, biopsy and X-rays. Abel's teaching regarding digital examination of the rectum should be followed: performing it with the left index finger, as the patient lies on the right side, because in this position the lower sigmoid falls into the pelvis and 2 in. to 3 in. more bowel are palpable through the rectal wall. I have repeatedly felt a recto-sigmoid tumour when, after an examination of the left side with the right index finger, nothing had been found.

May I here digress to mention that an inconspicuous carcinoma of the prostate may be associated with rectal ailments (e.g. piles or pain), or remoter symptoms (e.g. backache and sciatica). When a rectal condition is mainly in mind, there is a temptation to palpate the prostate cursorily and the diagnosis may be partial or missed.

The treatment of cancer of the rectum.—The superb abdomino-perineal resection was devised by Miles in 1907 and was practised by enthusiasts; but the perineal excision was favoured by the average general surgeon until the middle 'thirties because the mortality of the abdomino-perineal procedure was prohibitive, at 25% to 50%, whilst 90% of patients survived a perineal excision—although 70% recurred within five years.

Radium and carcinoma of the rectum.—In the period 1920 to 1935, Sir Charles Gordon-Watson tested the effect of radium in carcinoma of the rectum, and when President in 1928, he described his radium results. His careful study revealed its usefulness in low and medium grade anal cancers, but not for those of the rectum. To-day radium and deep X-ray therapy have a real but limited use in the very early and in the inoperable *anal* carcinomata.

The perineo-abdominal excision.—In 1932 W. B. Gabriel began his successful perineo-abdominal removal of the rectum. He tells me that he has done this operation 1,169 times, with a total mortality of 9·4%, but in his last 570 patients only 5·4% have died. His complete, fully analysed, five-year follow-up of all these patients is a pattern for surgeons.

The synchronous combined excision of the rectum.—In 1939, a new era was opened when Lloyd-Davies described the lithotomy-Trendelenburg position for excision of the rectum, thereby enabling two surgeons to operate together.

The operability rate of carcinoma of the rectum has increased to over 90% and the mortality has fallen to 10% even when taking into account the more extensive procedures now done and the older patients treated, some of whom are octogenarians. Miles' upper age limit was 65 years. This success is also in part due to the modern use of antibiotics, blood transfusions, better anæsthetics and improved techniques.

In 1942 Abel described the life-saving value of meticulous operative technique and of our debt to living surgeons (unpublished). He is the champion exponent of the Miles abdomino-perineal excision and many of us are indebted to him for instructing us how to

do it. In Britain and America he has pioneered stainless steel as the best surgical suture material; it has won general acceptance. Gabriel speaks highly of it for closing the abdomen.

The colon.—The wartime injuries of the colon were reviewed by Lockhart-Mummery in 1940, in the last of his several terms as our President. Proctology and this Section indeed owe much to him. He pioneered the sigmoidoscope from 1904 and continued until recently in thought, writing and practice. We salute him gratefully, he is our oldest living President.

Diverticulitis.—In 1952 Lloyd-Davies gave us a balanced statement on diverticulitis. He advises operation for it when blood is seen in the bowel, for in 6 of 9 of his patients with diverticulitis and bleeding, a tumour was present. Again when a colostomy is needed for an emergency due to diverticulitis, it should be a transverse one, and it should not be closed until the bowel affected has been resected. Finally he says that "despite the laborious and sometimes Herculean nature of these operations, the results have been most gratifying"; therefore he urges earlier resection of bowel affected with diverticulitis. I am doing this operation much oftener nowadays and patients derive considerable relief from a wide excision and accurate end-to-end anastomosis.

Ulcerative colitis.—Ulcerative colitis has become a frequent problem but before the war a bold approach was pioneered by Corbett (1945). He proved the great benefit of an ileostomy and later of a colectomy. When President he said "Good results from surgical treatment depend not only on the care of operative details but also on the pre- and post-operative management. The latter implies close co-operation between physician and surgeon". The past decade has confirmed his work, which has brought much relief to patients and is almost standard practice for this intransigent and not infrequent condition. Like a colostomy, all surgeons must be able to do an ileostomy skilfully for it is often an emergency. Later a partial or a complete colectomy, perhaps with the rectum, may be required. The possibilities of a carcinoma developing must always influence our decision.

Tumours of the colon.—Tumours of the colon have been dealt with by three outstanding Presidents. In 1927, J. P. Lockhart-Mummery spoke on the simple tumours, emphasizing their potential malignancy.

In 1931, Lionel Norbury pointed out the frequency of multiple primary carcinomata of the rectum and colon, and he alerted us to palpating the entire large bowel at operation.

In 1935, Sir Gordon Gordon-Taylor delivered a Presidential Address on "The complex and complicated in the surgery of the large bowel". His challenge to surgeons still resounds after twenty years. Although anti-shock measures and bacteriostatics were not available he achieved a notable success in cases which included single and multiple colonic growths involving every abdominal viscus, as well as, in some cases, resecting co-existing tumours of the stomach and small intestine. In spite of the growing enlightenment of medical men and the laity, the greater accuracy of X-ray and instrumental diagnosis, surgeons continue to be confronted with these multiple conditions. The diagnosis may be a problem, such as differentiating a carcinoma from a diverticulitic granuloma, after which a procedure requiring three to four hours' painstaking concentration may be necessary. The possibility of staged operations should be considered.

A total colectomy is becoming more and more advisable as was shown by Dickson Wright in his dissertation in 1949 (unpublished). A procto-ileostomy is now known to be compatible with a comfortable and gainful life. May I add that in defining the chief blood vessels of the colon I have found the technical experience gained from the dissection of varicose veins to be an invaluable apprenticeship.

The small intestine.—The territory of the "fundamentalists", as proctologists sometimes style themselves, has steadily extended. The ileum was included in 1946 when Crook reviewed the non-specific granulomata of Crohn's disease, showing how like ulcerative colitis it may affect both the large and small intestine.

In 1949 Michael Smyth when President ascended farther and described gastro-colic fistulæ. We also recall that Abel's Jacksonian prize essay was on the œsophagus, thus the ambit of the proctologist is the digestive tract, and this underlines our assignment as general surgeons with special knowledge, training and experience in the alimentary canal.

The first Charter of Proctology was drawn by Professor Grey Turner, to whom we are also indebted for his pioneer work on œsophagectomy, reconstruction of the rectum and transplantation of the ureters. The seven points he made were, the importance of proctological work, the necessity for a wide general training for the proctologist, the application of the general principles of surgery to the rectum and colon, the examination of the patient, what proctology can offer to greater medicine, reproaches it must try to remove, common ground for further study and the teaching of proctology. These have largely been established.

But we are living in a vastly changed world. The common achievements of surgery to-day were undreamt of thirty years ago, as also were the conflicting ideologies whose effects are tormenting many countries to-day. It is therefore fitting that we should add to

our Charter, and I would submit these four points: (1) *Our qualifications as surgeons*; (2) *Judgment*; (3) *Surgical technique*; and (4) *Teamwork*.

With the overthrow of shock, hæmorrhage and sepsis, if the diagnosis, judgment, technique and teamwork are good, consistently successful results should be obtained. Failures and deaths should occasion a searching inquiry, self-examination and frank discussion with our colleagues.

The surgeon's qualifications.—All the Presidents of this Section stand out as general surgeons and men who have had experience in other lands.

Surgeons who undertake proctological work should continue in contact with the "growing edge" of general surgery throughout their career, being ready to adopt and adapt. Old truths should be respected, for what is new is not necessarily true.

The proctologist should be a general surgeon, and the general surgeon should be a proctologist, because the diseases of the intestine and anus are common, and found in every land.

In his Presidential Address to the British Medical Association in 1950 a physician, Sir Henry Cohen, said, "Surgeons must make themselves safe for surgery". Was he thinking of the extensive operations that are possible to-day? I interpret him to require of us fitness, physically, mentally and spiritually with a knowledge of the purpose of life. It is a sobering fact that in this democratic age our *relationships with others are more important than our technical achievements.* Some of my mistakes have followed lack of experience and knowledge, others have been due to hurry, strained relationships at home and in hospital, the time of the day and the state of my health. How can we make ourselves safe for our patients?

(1) Physically we should be fit, then we can ask our patients to aim at the level of health which we ourselves enjoy. Some operations require two to seven hours concentrated effort. Fitness sustains this. Further, operation lists should be planned where a patient surgically fit, meets a fit surgeon and an adequate team. Relaxation is the counterpart of activity; using the seventh day as designed, gives us seven weeks' holiday a year. Could this be a key for a profession in which concentration is more practised than relaxation?

(2) *Mental fitness.*—We need adequate sleep, for fatigue contributes to forgetfulness, errors of judgment and irritable reactions. Good literature should be on our desks and at the bedside, the Book of Books being amongst them. Reading aloud and memorizing poetry is refreshing. Studying professional journals is essential. Participation in the activities of a Society like this, as well as regular travel, is necessary. They keep us receptive.

(3) *Spiritual fitness.*—Sir Russell Brain (1953) said, "Medicine alone took as its province the whole man", i.e. body, mind and spirit. Traditionally British medicine started on this total basis, but we have gradually focused on disease, leaving the man himself to others.

We recognize the effects of illness on the personality but must also know how personal disharmony, bitterness, and defeat may underlie a physical manifestation in a patient and possibly in ourselves.

By identifying ourselves with a Christian ideology, we lay the foundations of a right judgment with regard to patients, life and living.

We should also be citizens as well as surgeons, conscious of our country's direction, for our fellow-men must not be restored to health merely to be pawns in an ideological struggle for world domination, a conflict which is plain to see in homes, hospitals, industry and governments. We need an ideology, otherwise we become confused and expediency takes the place of principle which in the long run fails our patients, our fellows, ourselves and our country.

(4) *Judgment.*—An accurate diagnosis will always be expected of us, its correctness increases in proportion to the time we spend at the bedside, as opposed to the theatre; it is continued at the operation. We must know our "living" pathology. It is better to look *and* see than to wait and see. I have had to accept reproaches for omitting a laparotomy in an early case when time has confirmed the initial suspicion.

Judgment is composed of several factors, some being unavailable to us, but first we gather the maximum facts, evaluate and integrate them. The immediate and long-term plan should take into account the circumstances of each patient and his family. In septuagenarians and octogenarians especially with the radical operations at our disposal, can we decide on a wholly scientific basis what to do for them? I for my part need a wisdom higher than my own.

Technique.—Norman Tanner said with regard to his resections of the œsophagus in older patients, that they died of his technical errors and not of their disease. This has been my experience with major rectal and colon operations. As far as possible, our operations are made pattern procedures, departures being made only with the complex and the complicated. So long as we observe sound surgical principles there is wide scope for individual creative technique.

Besides the operation, pre-operative and post-operative care looms large in our planning to-day. Consideration is given to *thrombosis* in its various forms, because this is as yet an unsolved problem. At present, our best treatment is its prevention by never allowing the blood stream to become slow, particularly during the two to three hours immediately after an operation. The simple expedient of raising the foot of the bed of every unconscious adult assists this and saves lives (Payling Wright *et al.*, 1952).

(4) *Teamwork.*—Unique teamwork has been demonstrated by the staff of St. Mark's Hospital for the past twenty years. The conjoined written and spoken words of these men in the search for Truth are a guide to surgeons everywhere. Gabriel's book testifies to it. Further examples of creative partnerships are the historic Miles-Abel association, Corbett's ulcerative colitis work and Butler and Hermon Taylor's co-operation at the London Hospital.

Nursing.—Good nursing is essential, and without it we are helpless. Nurses must have our constant appreciation and training; only so can they reach their highest efficiency. They are a vital part of the surgical team in the pre-operative, operative and post-operative service. We are all too prone to take their work for granted. Nurses, too, are often in short supply. Our teams now both medical, nursing and ancillary are composed of Commonwealth and non-Commonwealth members whose background, training and ability differ but they must be wisely led.

CONCLUSION

The individualistic surgeon is passing. Who would we have for surgery and proctology? Those who are citizens as well as surgeons, those able to make a diagnosis, form a judgment, and those capable of sustained perfect operating. They must lead a team whose loyalty is to their Nation and to their patient irrespective of class and colour. Finally, if we will think of finding and meeting the deepest needs of the patient, proctological or otherwise, and if we can realize that surgery can only achieve its highest destiny when linked with a worth-while ideology which embraces the world, then we have a Charter which will show us the way. For as we are, so is our Country!

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